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THE VALUE OF ELECTROLYTE, WATER AND ACID BASE BALANCE STUDIES IN RENAL DISEASE*

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DURING the last two years, at the Abington Memorial Hospital, both the Medical and Surgical Services, as well as the Laboratories of Clinical Pathology and Bio-Chemistry, have been particularly concerned with certain aspects of renal disease. We believe that the proper interpretation of some of our studies throws additional light upon several of the controversial aspects of nephritis and furnishes us with a more rational basis for their management. The purpose of this paper is to discuss some of these observations in the light of their clinical interpretation and their probable mechanism.

In the studies herein to be considered, we wish at the outset to acknowledge the debt we owe to our associates in the laboratory who not only furnished the chemical data, but also aided in every way with their advice and suggestions. Our thanks are due especially to Dr. John Eiman and Dr. Charles G. Grosscup, who have been of invaluable help in the preparation of this report.

In this discussion we are only concerned with vascular renal disease. Whether the primary lesion involves the capillaries of the glomerular tufts or the endothelium of the afferent arterioles to the glomerulæ the ultimate effect is to disturb normal renal function. It must be stated that when the outstanding changes are in the arterioles, the vascular disturbance is by no means confined to the kidneys, but is a systemic involvement of the smaller blood vessels with possibly greater and more obtrusive damage to the renal arterioles.

Furthermore, whether we are dealing with renal disturbances that are primarily glomerular, or merely arteriolar, the important fact to determine is whether the vascular changes in the kidney are actively progressive or whether the condition is essentially stationary. If, therefore, we group together for purposes of discussion the various renal lesions that have as their underlying factor vascular changes, the important things to evaluate are the degree of activity of the pathological process and the extent of impairment of renal function. Methods for determining the functional capacity of the kidneys have varied widely from time to time and are subject to different lines of approach in various clinics. This has brought about varied interpretations of laboratory data. Whatever methods are followed, the object in view is to determine the functional capacity of the kidneys and to try to estimate their factor of safety and how great a burden they can carry.

There is no symptom-complex associated with renal insufficiency that is of more clinical importance or that has occasioned more experimental effort than the phenomena which have been called uremia. A number of clinical manifestations of totally different origin have heretofore been grouped under this general term. It is important, therefore, to agree upon what is meant by uremia. According to Volhard, there are three conditions that give rise to symptoms designated as uremia, but only those which are dependent upon renal insufficiency should be designated as true uremia; the others can be more appropriately termed extra-renal or pseudo-

*From the Medical Wards and Laboratory of Bio-Chemistry of the Abington Memorial Hospital. Read before the St. Paul Clinical Club, May 2, 1936.

uremia. Under this heading may be grouped: acute eclamptic uremia, and those cerebral circulatory phenomena associated with primary hypertension. In the first instance, the symptoms seem to be due to increased intracranial pressure due to cerebral edema. In the second, it is presumed that the manifestations are the outcome of cerebral ischemia, the result of the vaso-constriction that is held to be an active mechanism in hypertension.

Our concern is not with these types but only with true uremia, which results from renal insufficiency. Without presuming to discuss the pathogenesis of uremia, it may be stated with reasonable certainty that the clinical manifestations of true uremia are correlated in some way with retention in the blood of waste products that should have been eliminated by the kidneys. The most serious and lethal manifestation of true uremia is uremic coma and we have had the opportunity to study a number of cases that have led us to certain conclusions in regard to this important symptom. We have yet to observe a case of uremic coma without the presence of definite acidosis. This has led us to adopt the axiom, "No uremic coma without acidosis."

It would seem appropriate at this point to discuss, briefly, the mechanism by which this is brought about. It is true that most individuals with kidney insufficiency who become uremic present an elevation of the blood urea. This fact alone cannot be taken as indicative of impending coma since we have observed, along with others, many patients in whom the blood urea nitrogen reaches excessive levels, even as high as 300 mgms. but who show no evidences of coma. Attention should be called to the fact that, although most patients who have evidences of renal insufficiency that may lead to uremia show an elevation of blood urea nitrogen, there is a definite group of individuals who, even when other evidences point clearly to renal damage, such as a lack of ability to concentrate, markedly diminished phenolsulphonphthalein output and a decreased urea clearance, present a blood urea nitrogen which is only slightly elevated. When this situation arises, a study should be made to determine the functional efficiency of the liver, since numerous observations would lead us to believe that failure of the blood urea nitrogen to rise definitely in the face of other evidences of renal dysfunction is indicative of extensive liver damage. It has been repeatedly shown that serious parenchymal liver changes interfere with the production of urea. In cases of this kind, even when the blood urea nitrogen is not elevated, a definite and considerable rise in the total non-protein nitrogen is still present. The difference between these two figures, non-protein nitrogen and blood urea nitrogen, which is normally about 15 mgms., becomes considerably greater under such circumstances.

The blood urea level in nephritis is somewhat analogous to the blood sugar level in the diabetic. The diabetic does not die of a high blood sugar, neither does the nephritic succumb to a high blood urea. The diabetic becomes comatose because of the acidosis which is due to ketosis, low total base, or both. The comatose uremic patient does not die of a high blood urea but of acidosis that results from the inability of the damaged kidney to eliminate properly inorganic phosphates and sulphates which, as a consequence, accumulate in the blood and disturb the normal acid-base ratio. It should be added here that many patients with uremic coma are encountered who have failed to take an adequate amount of food, especially carbohydrates, and, therefore, there may be and often is the super-added factor of starvation ketosis.

The determination of the CO_2 combining power of the plasma is the most valuable index of the degree of acidosis. In acidosis of renal origin, besides low CO_2 capacity, there occurs an associated elevation of the blood inorganic phosphates. The same applies to the sulphates but, unfortunately, satisfactory methods for this determination are lacking. Furthermore, the elevation of the phosphorus gives a rapid and fairly reliable index of the impending danger of coma in renal insufficiency. When low plasma CO_2 is found with a normal blood phosphorus, look for conditions other than renal as the cause of the coma.

It seems likely that the acidosis of renal origin is brought about by an increase in sulphates and phosphates and, to some extent, organic acids, in conjunction with a lowering of total base level due to electrolyte loss through the kidney, caused principally by the inability of the functionally impaired kidney to metabolize ammonia for the neutralization of acid metabolites. Since nearly

TABLE I (CASE 1)

1936	4/27	4/28	4/29	4/30	5/1	5/8	5/18
BLOOD CHEMISTRY:							
Urea Nitrogen	210	160	160	140	130	88	67
Creatinine	10						
Plasma Chlorides	600	530	570	538	510	520	600
Plasma CO_2	18	33	57	48	39	59	38
Phosphorus	7.4						
Total Base	127	138					
BLOOD PRESSURE	110		152	140	120	134	142
	70		100	90	80	100	98
INTAKE							
Water c.c.	3,800	1,800	2,700	2,100	1,350	2,500	3,500
Sodium Lactate gms.	44.6						
NaCl gms.		4.5		5±	5±	12±	12±
$NaHCO_3$ gms.			12	12	12	12	12
Glucose gms.	200	100	125				
	Intravenously Incontinent						
URINE							
Amount 24 hours c.c.			1,490	2,380		2,510	1,390
NaCl 24 hours gms.			3.1	4.2		3.6	5.28
NaCl concentration			.2	.17		.14	.38
Specific Gravity	1.009			1.005			
Albumin	++++			+++			
Pus	+++			+			
Casts	0			0			
Urea Clearance							8.6
P. S. P. 2 hours						0	

all acids must be neutralized by ammonia or a fixed base before elimination by the kidneys, if the ability of the kidneys to supply the normal amount of ammonia is lessened, the burden must fall upon the fixed bases of the body fluids such as sodium and potassium. The loss of these valuable basic constituents brings about, therefore, a reduction in the total level of the base.

When the total base is lowered, dehydration occurs and both salt and water are required by such individuals. In order to illustrate the points under discussion, a patient (Case 1) who has come under our observation may be cited briefly.

Case 1.—A woman, aged thirty-seven, presented emaciation, pallor, confusion and various delusions. Her breathing was of the Kussmaul type; there was evidence of left ventricular hypertrophy and relative mitral insufficiency. She was incontinent with a scanty output. The abdomen was negative, there was no loss of voluntary motion and no abnormal reflexes were present. It is worthy of note that the systolic blood pressure was only 100, diastolic 70. The blood showed a moderate secondary anemia. The urine had a specific gravity that varied between 1.005 and 1.009; albumin was present in definite amounts along with white blood cells and casts. The blood urea nitrogen was 210; blood sugar 190; plasma chlorides 600; total base 127; the phosphorus was up to 7.4 and the plasma CO_2 down to 18. Lumbar puncture was done and the spinal fluid was found clear and not under pressure. She was given, intravenously, 44.6 gms. sodium lactate, 200 gms. glucose and 3,800 c.c. water. The next day she was less confused. The blood chemistry was as follows: blood urea nitrogen 160; plasma chlorides 530; CO_2 33; total base 138. She was given, intravenously, NaCl 4.5 gms., glucose 100 gms. and 1,800 c.c. water. The third day she was rational and continent. Her blood pressure was 152/100 and her urine output 1,490 c.c. The blood chemistry was as follows: blood urea nitrogen 160; chlorides 570; CO_2 57. From the fourth day on she was given daily, by mouth, 12 gms. sodium bicarbonate and from 5 to 12 gms. of sodium chloride.

It should be noted that as her blood pressure rose from 110/70 to 152/100, there was an improvement in the urinary output. Twenty days after admission she was in excellent condition physically and mentally; her blood pressure was 142/98 and the blood chemistry as follows: blood urea nitrogen 67; chlorides 600; CO_2 38; urea clearance 8.6 per cent; phenolsulphonphthalein 0.

When she was admitted, it was realized that because of high phosphorus and a low CO_2, she was in a state of impending coma. Since the possibility of diabetes could be excluded and there were undeniable evidences of chronic renal disease present, it was reasonable to suppose that the coma was of uremic origin.

By the use of sodium bicarbonate by mouth, but particularly, 5 per cent glucose and one-sixth molar sodium lactate solution intravenously, along with several small blood transfusions, it was possible not only to avert profound coma but to standardize gradually

this individual and to bring her blood chemistry back to approximately normal equilibrium. She was discharged markedly improved and was directed to take daily 3 gms. of sodium bicarbonate.

Since then she has been readmitted several times. On a subsequent admission, she gave a history of having stopped her sodium bicarbonate. She was semicomatose with a low blood pressure, her urine with a specific gravity of around 1.005, containing a heavy cloud of albumin and many casts. Her blood urea nitrogen was 210; chlorides, 546; blood phosphates, 6.2, and CO_2 down to 14. On each admission, she was again given glucose and sodium lactate intravenously and within twenty-four hours, she gradually began to come out of her coma, her blood urea nitrogen came down, CO_2 rose and her phosphorus was considerably reduced.

It is interesting that in this case the impairment of renal function and subsequent uremia always went hand in hand with a marked lowering of the blood pressure. Since adequate renal function depends largely upon the maintenance of a proper blood flow through the kidneys and adequate pressure in the glomerular tufts, it is reasonable to assume that when the systemic blood pressure falls below a certain level, filtration through the glomeruli is markedly decreased. It has been shown that the pressure in the glomeruli is normally unusually high, about the same or 20 per cent less than the carotid pressure.

In connection with the question of uremia, it is appropriate to call attention to a certain group of cases that, in the past, have not infrequently been mistaken for uremia. When excessive loss of salt and water results from vomiting, diarrhea or excessive sweating, dehydration and hypochloremia result. When the plasma chlorides fall to 500, the blood urea nitrogen is invariably increased and the lower the chlorides, the higher the blood urea nitrogen. In the vomiting cases, there is also an associated elevation of the plasma CO_2 combining power. We know now that the increase of blood urea and CO_2 (the latter indicating an increase in blood bicarbonate content) are part of a complicated mechanism designed to compensate for the loss of plasma chlorides and to maintain the osmotic pressure as near normal as possible.

From a practical standpoint, these cases of hypochloremia, as has been suggested, are of importance because of the ease with which they may be confused with uremia. When a patient with an irritable stomach or vomiting shows albumin in the urine and elevation of urea nitrogen, not infrequently a provisional diagnosis of nephritis is made. When, in such a case, in response to a widespread fallacy, salt in the diet is immediately restricted, the vomiting is promptly aggravated. As a result, the urea nitrogen will continue to rise to 100 or more and finally a state of coma may supervene. Then a diagnosis of uremia is certain to be made. Further salt restriction is apt to be instituted, accompanied by various measures such as sweats which increase dehydration. Unhappily, such an individual may lapse into coma and succumb. Had the blood chlorides been determined in such a case and had it been realized what actually occurred as the result of persistent vomiting, a proper diagnosis would have been reached. The case would have been recognized as one of hypochloremia with its associated alkalosis and azotemia. Proper salt and water therapy will promptly correct the condition, whereas the therapy commonly used only serves to make it worse. In cases of hypochloremia, unless there has been a pre-existing hypertension, the blood pressure is not elevated and may be considerably reduced. This is a differential point worthy of note.

Another important factor to be considered in the study and management of renal disease is the sodium chloride intake. Normally, there is a sodium chloride balance and the amount excreted in the urine is approximately equal to that ingested. It is of value in renal disease to know the total electrolyte concentration in the blood plasma because certain kidney cases have a tendency to store sodium chloride somewhere within the body in considerable quantities. The exact mechanism by which this is accomplished is far from being well understood.

It is a fact that many chronic renal cases have great difficulty in eliminating this stored sodium chloride. As a result, the plasma chlorides may rise to above 600. When this happens and the urea nitrogen is also elevated, serious alterations in osmotic pressure of the plasma occur. It is, therefore, of definite practical importance to bring the sodium chloride level down to normal limits in chronic renal cases even when the retention of sodium chlorides is not associated with edema. It has been found that many damaged kidneys cannot concentrate salt in the urine above 0.4 per cent, whereas the normal kidney

will concentrate up to 1.8 per cent or even 2 per cent. In view of this, it is advisable to limit the daily intake of sodium chloride in accordance with the amount that can be readily eliminated in order that the plasma chloride level will not be too high. The best way to determine this "salt tolerance" is to carry out salt balance studies over a period of several days. An effort should be made to see that the plasma chlorides remain somewhere around 550. A series of studies have shown that, in the average case, approximately 2.5 gms. of salt per day is a satisfactory maintenance dose.

In this connection, it should be recalled that the minimum requirement for a normal adult is only about 1.8 gms. per day, whereas the ordinary hospital "house diet" contains approximately 10 gms. This aspect of the subject is well illustrated by a case of chronic arteriolar nephrosclerosis which we had an opportunity to study over a long period of time. In this individual, the highest concentration of chlorides obtained in the urine was 0.4 per cent at a time when the plasma chlorides averaged 630 or more. It required fourteen days to eliminate the excess chlorides. This patient has since been maintained at a suitable level on 2.5 gms. per day.

Closely associated with the question of the sodium chloride balance is the problem of the restoration and maintenance of nephritics in a nearly normal water balance. Many nephritics are seen who are in a state of dehydration. However, there are many others in whom the opposite situation exists, namely, excessive water retention and edema. Both situations call for a careful consideration not only of the electrolyte level and water balance but also of the plasma protein concentration.

It is true that increased electrolytes, notably sodium chloride, are an important factor in holding water in the tissues and bringing about edema. On the other hand, it is useless to try to correct the electrolyte level without knowing whether the plasma proteins are above the edema level. If the total plasma proteins are below 5.3 per cent, the colloidal osmotic pressure is so altered that the retention of fluid in the interstitial tissue spaces results in edema. Under conditions of low plasma proteins, if water is forced with or without electrolytes, edema will occur. Therefore, in cases of this kind, either the plasma protein level must be raised by the increased ingestion of proteins or more effectively by transfusion, or the colloidal osmotic pressure must be restored temporarily by the injection of acacia solution.

Not only is it important to determine the amount of total plasma proteins present, but it is also of value to recognize any inversion of the normal serum albumin-serum globulin ratio. In view of the fact that the globulin molecule is larger than the albumin molecule, the effective osmotic pressure per unit mass of the former is less, which makes for the development of edema. When the plasma protein level is normal, or has been restored to normal, then the level of the solutes should also be regulated.

When these steps have been accomplished, water should be given to nephritics as freely as possible without embarrassing the circulation. This latter point should be given careful consideration in deciding upon the amount of water to give a nephritic. In nephritics, especially the chronic ones with hypertension, it is essential to maintain the integrity of the myocardium. The ingestion of excessive amounts of fluid, especially if administered rapidly, may raise the blood volume to the point of throwing an overload on a weakened heart muscle, which may have disastrous results. On the other hand, since it is of fundamental importance to eliminate as much of the metabolites as possible through the damaged kidneys and, thereby, prevent their accumulation, the maximum amount of water should be administered. It is unnecessary to draw attention to the well known fact that special care must be exercised in the giving of sodium chloride and water when edema or effusion exist.

Some of these problems are illustrated in the following patient:

Case 2.—A laborer, aged forty-eight, has been under observation for the past two years suffering from a definite chronic arteriolar nephrosclerosis of long standing. The condition was non-progressive and he was symptom-free for years. On admission, he presented unmistakable evidence of chronic arteriolar nephritis and prostate hypertophy with an associated cystitis and obstruction. Within a few days he became drowsy. At this time, his blood urea nitrogen was 276, his chlorides 490. His blood plasma, CO_2, had dropped to 29, and the phosphorus had risen to 8.2. His urea clearance was 8 per cent, and P.S.P. output, 0. For the next few days, he had occasional vomiting and his drowsiness persisted. He had retention of urine which

TABLE II (CASE 2)

	1934								1935			
	10/30	11/1	11/7	11/16	11/26	11/27	12/1	12/11	1/20	2/20	4/4	11/22
BLOOD CHEMISTRY												
Urea Nitrogen	222	276	245	90	61	60	63	52	50	57	37	40
Creatinine			4.4				3.6					2.8
Sugar	125		109	85	73	79		90	92	82		
Plasma Chlorides	488	490	530	598	650	660	660	580	578	580	600	586
Pasma CO_2	35	29	46	49	37	44	51	47	51	52	46	50
Phosphorus	4.8	8.2	6.2	2.8	3.6	4	4	4	3.8	4.1	3.2	2.1
Plasma Proteins %				6.0				6.0				
URINE												
Amount 24 hrs. c.c.	1,200	1,550	2,200	3,400	4,100	4,100	4,500	2,950	1,790	1.970		1,155
NaCl 24 hrs. gm.						15.4	14.8	6.7	5.0	3.6		1.8
NaCl Concentration %						.37	.33	.22	.27	.18		.16
Specific Gravity	1.006	1.009	1.008	1.007	1.004		1.006	1.004				
Albumin	++	++	++	++	++							
RBC's	0	0	0	0	0							
Pus	+++	+++	+	++	±							
Casts	0	0	0	0	0			17	15			23
Urea Clearance %		8		4								6.4
P.S.P. % 2 hrs.		0		0								
INTAKE												
NaCl gms.	10±	10±	10±	10±	10±	2.5	2.5	2.5	2.5	2.5+	2.5+	2.5
$NHCO_3$ gms.	0	4	4	4	0	1	1	1	1	1±	1±	1±
		Start		last day		start						
Blood Pressure	116		110	118	120	120	116	104				
	68		60	82	84	80	68	60				
REMARKS		Tranfusion							Prostatic Resection	Returned to work	Working	

required a gradual decompression. His blood urea nitrogen continued above 200 and his blood phosphorus remained high, between 7.8 and 6.2, until the decompression was completed. As his blood urea nitrogen came down to 90, his plasma CO_2 went up to 49, and the phosphorus dropped to 2.8, his drowsiness disappeared.

Shortly after admission, when his chlorides were low (488), he was placed on a house diet averaging 10 gms. of sodium chloride per day. On this intake, his plasma chlorides gradually rose until about one month after admission they reached 660. He was then placed on daily sodium chloride intake of 2.5 gms. Notwithstanding this low intake, it took about fourteen days to reduce his plasma chlorides to 580.

In order to combat the tendency to acidosis, he was given daily by mouth, from 1 to 4 gms. of sodium bicarbonate.

When he was discharged, he was taking daily, 1 gm. sodium bicarbonate, a low salt diet with moderate restriction of protein intake and forced fluids in view of the fact that he had no edema and showed no signs of circulatory embarrassment.

A month later his condition was sufficiently improved to warrant a transurethral resection of the prostate under local anesthesia. His recovery was prompt and on his discharge, a few weeks later, the patient was voiding without trouble and his blood urea nitrogen was 57; plasma chlorides, 580; CO_2, 52; phosphorus, 4.1; urea clearance, 15.

Subsequent reports covering a period of about eighteen months indicate that his general condition continues to be satisfactory and he is able to continue active physical work. His laboratory findings recently were as follows: blood urea nitrogen, 40; creatinine, 2.8; plasma chlorides, 586, CO_2, 50; phosphorus, 2:1; urea clearance, 23, and P. S. P., 6.4.

This case illustrates the important fact that, in spite of his chronic nephropathy, he remained in good condition for years until the superadded urinary tract obstruction, with infection, occurred. This, as so often happens, precipitated the uremic coma which was impending as indi-

cated by his drowsiness, the high blood phosphorus and the falling plasma CO_2. By careful regulation of his sodium chloride, water and protein intake, and combating his acidosis and at the same time relieving at first temporarily and later permanently, the urethral obstruction, it was possible, gradually, to improve his kidney function to the point where he was a reasonable operative risk and, furthermore, to get him in such condition that he was able to return to useful occupation. It is such a case as this that shows what may be done along the lines of standardizing nephritics provided the nephritic is one in whom the process is not progressive, and the initial inflammatory lesion has become arrested.

A different type of case is presented by the following:

Case 3.—A young woman, aged thirty-two, was admitted to the hospital with a diagnosis of acute proliferative arteriolar nephropathy with beginning hypertension. Her physical examination, urinary findings, blood chemistry and renal function studies amply confirmed this diagnosis. Her eye grounds showed angiosclerosis of the retinal vessels. Her tonsils were found to be diseased and cultures from the tonsils and stools showed streptococcus viridans and hemolytic streptococci. As the only demonstrable focus of infection that could be found was her tonsils, these were removed in the hope that, in this way, her renal condition could be improved and the process checked. This, unfortunately, failed to be the case, as during her stay in the hospital her urinary findings did not improve. On the contrary, evidence of progressive renal insufficiency occurred.

Unlike the patients previously referred to, it was impossible to standardize this patient because the vascular lesions of the kidneys were progressive even when a focus of infection was removed. She developed a severe toxic purpura and died from cerebral and pulmonary edema.

Here is an instance of an individual in whom, evidently, the fundamental cause for her malignant proliferative nephro-arteriolitis was a streptococcus infection which brought about progressive uncontrollable renal changes.

In the case of this woman, the terminal nervous phenomena were not the result of true uremia—no stupor or coma and no acidosis. On the other hand, the disturbances of her blood chemistry, increase of solute concentration, evidently induced an increase in osmotic pressure of the body fluids that led to the development of cerebral and pulmonary edema. The mechanism by which such changes occur is certainly not well understood and is not properly a part of such a discussion as this.

From a practical standpoint, the various studies we have carried out have led us to believe that, discouraging as the management of the chronic vascular renal case may be, the situation is by no means hopeless in certain selected instances.

In cases of impending coma, besides regulating water and electrolyte balance, the important problem is the proper administration of alkali. If the plasma CO_2 is 20 or under, sodium lactate in ⅙ molar solution, administered intravenously, is the alkali of choice (60 c.c. of ⅙ M. solution per kil. of body weight will raise the plasma CO_2 combining power 30 volumes per cent). In cases in which the CO_2 is above 20 and the patient is able to take liquids by mouth, the administration of sodium bicarbonate by that route will often suffice (0.78 gms. per kil. of body weight will raise the CO_2 combining power 30 volumes per cent). If such treatment is promptly instituted, it will not infrequently be possible to relieve or prevent uremic coma. The individuals who are suspected of suffering from the so-called wet brain should be given the benefit of a lumbar puncture, since the relief of the spinal pressure will often bring about considerable improvement, especially if it is repeated sufficiently often. In all instances, where patients have been on inadequate diets and are unconscious or vomiting, in order to combat and prevent starvation acidosis, an ample supply of glucose should be administered in a 5 per cent solution.

The cases that fall in the category of the man with chronic nephrosclerosis offer an excellent opportunity for standardization, if careful and sufficiently prolonged studies are carried out. In the standardization of such a nephritic, a procedure should be followed not unlike that in vogue for the standardization of the diabetic. The sodium chloride intake and output must be carefully observed. The safe daily requirements of salt can be determined with considerable accuracy. The same thing must apply to the administration of a base, such as sodium bicarbonate or lactate, if the necessity for it exists. Protein intake is of great importance. The error is frequently made of restricting the nephritic too much in protein intake, thereby bringing about

a negative nitrogen balance. Excessively low protein diets, 28 to 40 gms., may be justified for short periods of time. Most patients should receive close to one gram of protein per kilo of body weight, and the ordinary adult requires not less than 60 gms. per day. When the sodium chloride, protein and base requirements have been properly determined and there is no noteworthy edema, the water intake should be the maximum the patient can take without causing circulatory embarrassment.

Our conclusions may be briefly summarized as follows:

1. No true uremic coma without acidosis.
2. There is no relationship between uremic coma and the height of the blood urea.
3. In acidosis of renal origin, there is decrease in the plasma CO_2 combining power plus increase in inorganic phosphates and sulphates of the blood. There may be associated reduction of total base and "starvation ketosis."
4. When systemic blood pressure in a chonic nephritic falls to a level below the effective glomerular filtration pressure, renal function will be markedly impaired and uremic coma may result.
5. Cases of hypochloremia with high blood urea often are mistaken for true uremia. The hyperazotemia and increased plasma CO_2 are part of a compensatory mechanism for the maintenance of osmotic pressure of body fluids. Unless hypertension pre-exists, these cases will show no elevation of blood pressure; on the contrary, there is often a lowered blood pressure.
6. Chronicly diseased kidneys eliminate sodium chloride only in low concentration. Even when no edema is present, it is important to regulate sodium chloride intake so as to preserve a normal electrolyte level in order to maintain a normal osmotic pressure.
7. Water intake in a nephritic must be regulated in accordance with the electrolyte level, the quantity of total plasma proteins present, and the efficiency of the myocardium. The maximum amount possible under the existing conditions should be given.
8. The chronic vascular nephritic, in whom the condition is no longer progressive, may live in reasonable comfort if properly standardized and maintained in water, sodium chloride, alkali, and protein balances. In the more acute progressive cases, this cannot be done successfully.

In the foregoing, an effort has been made to collect and to systematize some observations on the study and management of the vascular forms of chronic renal disease. This has been done in the belief that certain definite facts have been demonstrated and conclusions reached which may prove an aid to the clinician at the bedside in the more successful management of this group of nephropathies.

HISTOLOGY OF THE PINEAL GLAND AND ITS PROBABLE PHYSIOLOGIC FUNCTION*

ADOLPH M. HANSON, M.D.

Faribault, Minnesota

IT is with grateful acknowledgment of Dr. Percival Bailey's gracious permission that I am privileged to present the accompanying illustrations of the cellular structure of the pineal body as first studied and brought out by Hortega.†

The pineal gland received its name in 1644 from Thomas Willis (1621-1675) in much the same manner as it received a former name, "conarium" ("Pineus" means "pine cone").

In Thomas Gibson's "Anatomy of human bodies epitomized" (1682), it is stated: "The glandula pinealis, or penis, because it representeth the pine-nut of a man's yard. It is seated in the beginning of that pipe by which the third and fourth ventricles are united." How appropriately it has been named "the penis of the brain" is now becoming apparent. According to Galen (131-201; A. D.), it is a secreting gland.

This small gland, that median eye of our

*Read before the meeting of the Southern Minnesota Medical Association, Albert Lea, Minnesota, August 31, 1936.

†The illustrations were secured by photostatic copies of Bailey's original article through the coöperation and courtesy of Dr. Oliver Kamm and Parke, Davis and Company.

arachnoid ancestors, from being the lone all-seeing eye of the past seems to be the hidden, back-seat driver of human destinies, the nigger in the woodpile of human affairs and conduct, that in some way, for good or for evil, may influence the acts of the ego in the flesh more than any other one gland. It was laid down with the foundations of the face and, therefore, was most likely the very first tissue, or one of the first tissues, to take on the function of internal secretion. It arises behind the superior commissure from the posterior surface of the interbrain as a small tube. In cyclostomes, many fishes and reptiles, it extends through the cranium to the skin on the dorsal surface of the head, resembles the eye in structure, and seems to function as an organ for perception of light. For this reason it has been called the median or pineal eye. Special light-percipient cells have been found in the forebrain and throughout the spinal cord in the Amphioxus and in ganglia of certain worms. The pineal eyes originally seem to be paired as in adult cyclostomes, and in some reptiles they appear one before the other. In some classes of vertebrates the position and innervation indicate that it is the anterior or sometimes the posterior organ which is present. The anterior body projects its nerve fibres into the nucleus habenulæ, while those from the posterior go to the posterior commissure and the tectum opticum. It is said that even the degenerated organs in vertebrates, in cyclostomes, some reptiles and fishes, are still capable of some perception of light. The structure is much simpler than that of the retina, but in some, rods and cones have been found. As evolution progresses one or the other of the two pineal eyes disappears, so that, while the organ was originally paired, one only remains. The blepharoplasten,

Fig. 1. Parenchymal cells of the human epiphysis, impregnated by a specific method. Some cells are much more heavily impregnated than others. A, parenchymal cells; B, tuberous marginal extremities; C (just below the letter B, in the middle zone of the section), interlobar space filled with end bulbs; D (extreme lower right), vessel surrounded by end bulbs (after Hortega).

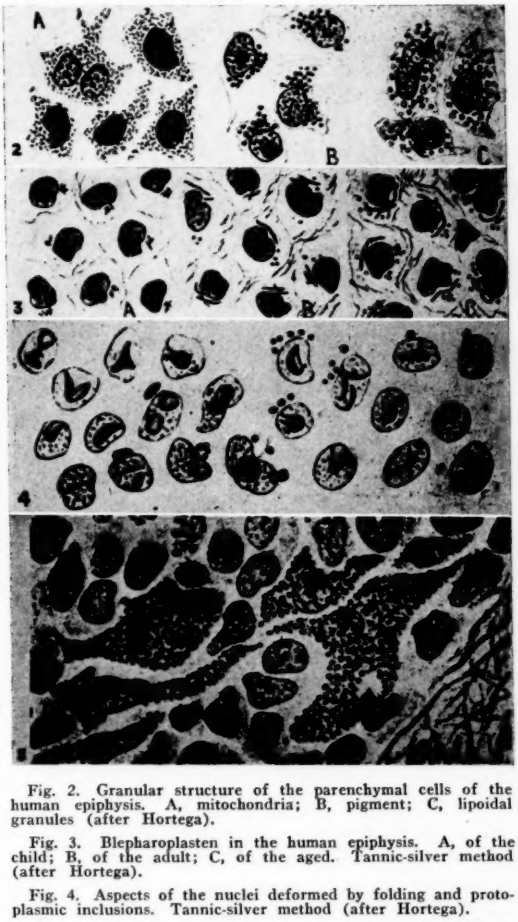

Fig. 2. Granular structure of the parenchymal cells of the human epiphysis. A, mitochondria; B, pigment; C, lipoidal granules (after Hortega).

Fig. 3. Blepharoplasten in the human epiphysis. A, of the child; B, of the adult; C, of the aged. Tannic-silver method (after Hortega).

Fig. 4. Aspects of the nuclei deformed by folding and protoplasmic inclusions. Tannic-silver method (after Hortega).

Fig. 5. Granular neuroglial cells in the human epiphysis. Silver-carbonate method (after Hortega).

found in the parenchymal cells of the bovine and human pineal bodies, suggest the primitive blepharoplast, a minute mass of chromatin formed from the nucleus in certain protoza, or forming the base of a flagellum acting as the center for the movement of the organism. Because phylogenetic study has revealed the pineal as the primitive eye, many have scoffed at the idea that it could in any wise carry on the function of internal secretion. Here again we find that it may even be worldly wise to "hope all things; believe all things; try all things and retain that which is good."

The pineal body in the human being seems fully developed some time in the first year of life. Early in life the pineal gland contains a variety of cells. The blood vessels invade the gland at about the fourth month of fetal life and septa begin to grow into it and are well marked at eight or nine years of age. This lobulation of the gland may or may not become complete in the adult.

Bailey stated: "In the human pineal body, aside from the ependymal cells lining the recessus and cavum pinale and the connective tissue, there are found only two cellular types, the pineal parenchyma and the neuroglia. True nerve cells have possibly been identified in a few rare instances." From this statement it will be understood that the pineal gland—that is, that structure contained within its capsule, contains two distinct types of cells and two only.

I base my statements concerning what seem to me the principal considerations from the histologic viewpoint, on Bailey's scholarly discussion of the subject in which he, in turn, gave due credit to Achücarro and Sacristän, Walter, Josephy and del Rio Hortega. It is because of the work of del Rio Hortega, in 1922, and his special method of impregnation, that the structure of the pineal parenchymal cell was finally demonstrated. All of the cells have tubular processes that terminate in end-bulbs near connective tissue and surounding blood vessels (Fig. 1). The pigments in the cells are intensely argentophilic (Fig. 2, A and B). Spherules giving a lipoidal reaction are found; they are illustrated in Figure 2 C. In addition to the argentophilic granules and lipoidal spherules, each cell contains one or more rods in a group near the nucleus that take the stain of blepharoplasten, such as is seen in ependymal cells (Fig. 3). There is also a Golgi apparatus. A similar structure is seen in nerve cells in the cerebral cortex and posterior columns of the cord. These cells have a short axone that breaks up into very numerous branches but does not emerge from the grey matter and its seeming purpose is that of bringing neighboring cells into relation with one another.

The neuroglial cells resemble those of the brain in many respects. A few gliosomes are found in these cells.

Hortega identified granules, stained by Achücarro's method, that he believed to be secretory granules. Nagéotte believe that gliosomes were, also, secretory granules.

The nucleus is supposed to play a part in secretion and such nuclei have been found to have a fold in their membranes. Some may be artefacts, while others are attributable to definite protoplasmic inclusions. Such nuclei are found in the parenchymal cells of the pineal (Fig. 4). The secretory granules of the parenchymal cell are probably blepharoplasten. The granules in the neuroglial cells, as brought out by the silver-chromate method, are presented in Figure 5.

At the time when Bailey wrote his article, in 1932, in which he briefly summarized the consensus of the best opinion concerning the function of the pineal body, he ended with this sentence: "It is probable that the pineal body of man is a rudimentary structure without function." From what was said in the article it will be sensed that the best minds, still in a lingering fashion, thought that it was perhaps probable that this little gland might be of some significance. However, I doubt if they thought that it might be the nigger in the woodpile of human affairs and conduct and, perhaps, the back-seat driver of human destinies.

René Descartes, the father of the postulate, "I think, therefore I am" (cogito ergo sum), and one of the world's greatest contributors to philosophy, physical science and mathematics, even though he died in 1650, thought the pineal body to be the seat of the soul. It now seems that it may influence the acts of that fundamental ego that is back of human personality more than any other one gland.

Early in 1927 the writer prepared twenty-seven beef and four sheep pineal extracts. Only one revealed a slight depressor effect. Dr. Leon-

ard G. Rowntree and his coworkers, using pineal extract (Hanson)* intraperitoneally, produced dwarfism and excessive sexual excitation and development in succeeding generations of white rats of the Wistar strain.

The really notable observation in this histologic study of the pineal gland, as revealed by del Rio Hortega, is the individual glandular character of each parenchymal cell with its tubular process and end-bulb. Allowing one's imagination to travel backward instead of forward, it is not difficult to visualize the earliest attempts of nature at secretion of any kind. Is it not likely that the first attempt was some simple cell like the parenchymal cell of the pineal? This thought leads me to the hypothesis that the pineal parenchymal cell is the most primitive cell of internal secretion in Man. The seeming function of pineal gland is a retarding or limiting effect in growth control and a primary sexual stimulation.

*A solution of a picrate precipitate from an acid extraction of bovine pineal glands from beef of average killing age. The pineal extract used in the study referred to was given to The Philadelphia Institute for Medical Research by the Hanson Research Laboratory under a grant from The Josiah Macy, Jr., Foundation of New York City.

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PRINCIPLES IN THE MEDICAL TREATMENT OF HEAD INJURIES*

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IN reference to the title, allow me to say that this paper will briefly cover the subject of the medical treatment of head injuries. The surgical management is not within its scope, and will be left to those better informed on this subject than myself.

However, this non-surgical group includes the great majority of cases of head injury. Because of the very efficient physiological methods at hand to reduce intracranial pressure, surgical decompression to control intracranial pressure is practiced only as a measure of last resort, and then only too often ends fatally by adding insult to the already traumatized brain. The patient may recover in spite of, but rarely because of, the decompression operation.

Please note that the above statement applies to the control of intracranial pressure and does not mean to include those cases of head injury with large depressed bone fragments, or the rapidly progressing subdural hemorrhages which are obvious surgical emergencies. Yet Dr. Temple Fay of Philadelphia, reporting on a group of 300 cases of severe head injuries, opened the skull in only fifteen cases, or 5 per cent. His mortality for the entire 300 cases was 18 per cent, but his mortality for the fifteen surgical cases was 80 per cent.

Simple depressed fractures or slight depressions of the inner table are not operative indications in themselves. They may be repaired at a later date for cosmetic reasons or in attempt to relieve persistent focal symptoms. The elevation of these small depressions of the skull will most likely not relieve the focal symptoms because these symptoms are generally due to contusion of the underlying brain itself.

The small linear fractures of the bony skull and often some of the large ones are in themselves of very little significance. In fact, the area of greatest damage to the brain may often be contra-coup in relation to the fracture as demonstrated by x-ray.

So, then, in the great majority of head injuries we are dealing with increased intracranial pressure due to edema or slow hemorrhage. These are the factors that must be combated by medical means along physiological principles if we are to aid in the recovery of our patient. Let me repeat, it is edema and slow hemorrhage that we

*Read before the Upper Mississippi Medical Society, Cass Lake, Minnesota, August 1, 1936.

must combat. In increased intracranial pressure we have three volumes to deal with. From left to right on the diagrammatic chart they are circulating arterial and venous blood, brain tissue itself, and the cerebrospinal fluid. All three are contained in a non-elastic chamber—the bony skull.

As the brain expands due to traumatic edema, the pressure within the skull is increased. This increased pressure is transmitted directly and immediately to the other volumes. Increased pressure is brought to bear on the volume normally occupied by the circulating arterial and venous blood. As a result, capillaries, veins, and the smaller arterioles are collapsed. So, then, in the end we are dealing with oxygen and nutritional starvation to the brain tissue due to lack of circulating blood.

The same pressure effect on the blood volume is produced by a subdural clot or hemorrhage into the subarachnoid, the volume space occupied by the cerebro-spinal fluid. It may seem hard to understand how any bleeding can take place in a closed non-elastic cavity filled with incompressible fluids. But we must remember that there is a difference in the pressure between the smallest arterioles and their accompanying veins. As a result venous sinuses are collapsed as hemorrhage causes increased intracranial pressure.

Brain cells are very sensitive cells. A few hours without proper oxygen or nutrition will cause their destruction, and they will never be regenerated again. That is why head injuries are a real emergency and demand the most prompt and thorough treatment. No physician should be content to simply place his patient in bed with an ice cap to the head and wait expectantly for nature to come to the rescue.

The application of the well filled, convex ice bag to the equally convex skull with an area of contact of but a few square inches should only be considered as therapy for the patient's anxious relatives. Very likely an ice collar about the neck to cool the blood in the carotid vessels would be more effective to the patient.

We can do very little to control the hemorrhage or the edema. But we can very definitely make room for the all-important space occupied by the circulating blood volume by changing the volume occupied by the cerebro-spinal fluid.

This may be accomplished by spinal drainage, the injection of hypertonic solutions intravenously, and by general dehydration. The principle of lowering the volume of cerebral spinal fluid by spinal puncture is obvious. The action of intravenous hypertonic solutions in lowering the volume of cerebro-spinal fluid is according to the

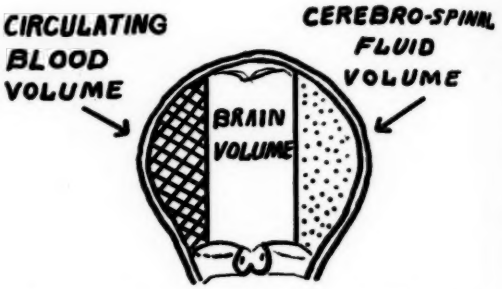

Fig. 1. A schematic diagram of the three important volume spaces within the skull.

principle of osmosis. We know that the spinal fluid is formed by the vascular fringes known as the choroid plexuses, located in all the ventricles. It is formed from blood plasma by a process of filtration through a membrane of characteristic permeability. The direction of flow is from the lateral ventricles through the foramina of Monroe into the third ventricle, then through the aqueduct of Sylvius and out through the foramina of Luschka and Magendie into the subarachnoid spaces. The volume is 100 to 150 c.c. The normal pressure varies between 60 and 120 mm. of water. With free drainage it may be formed at the rate of a liter a day.

Normally there is only 5 grams of glucose in the blood stream. By injecting 50 c.c. of a 50 per cent solution of glucose we put 25 more grams of glucose into the blood and so raise its osmotic pressure that the rate of formation of cerebral spinal fluid is greatly diminished. In fact, Weed has shown, by dye fixation tests, that by the use of hypertonic solutions intravenously the direction of flow of cerebro-spinal fluid can even be reversed, and the fluid will be reabsorbed by the choroid plexuses.

But we must remember that the use of hypertonic solutions is not a true dehydration. The cerebro-spinal fluid is only drawn back into the blood stream. No fluid has been lost from the body. The effect of this 25 grams of glucose, though very rapid, will only last about four hours and then must be repeated.

However, in the meantime we can establish

true general dehydration by limiting fluids, and by the use of saline cathartics given either by mouth, or as retention enemas. This is a slower process but absolutely permanent as long as dehydration is maintained. It is so effective in the control of the volume of cerebro-spinal fluid, that in a patient whose fluids have been limited to 600 c.c. per twenty-four hours for a few days, no spinal fluid can be obtained by dorsal spinal puncture with the patient in the reclining position.

Now you may ask, "Why go to all the trouble of intravenous solutions and dehydration when the same thing may be rapidly accomplished by repeated spinal punctures?"

It is believed that osmosis and dehydration simulate nature's way of controlling cerebro-spinal fluid volume, and that the more abrupt repeated spinal punctures add insult to the already traumatized brain.

Let me interrupt at this point to give one brief case history to illustrate the importance of dehydration therapy in brain injuries.

A large, robust, German farmer, aged thirty-five, was brought to our hospital with a severe head injury suffered when the tractor he was riding tipped over backward on him. He was unconscious for two days. After the initial period of shock was combated, he improved rapidly under intravenous glucose and dehydration therapy. He had been conscious and perfectly rational for six days with no focal symptoms of sensory or motor disturbance. There was no skull fracture visible on x-ray. On the seventh day he developed a persistent vomiting. We were afraid that he perhaps was losing too much fluid with this vomiting on top of his dehydration régime. So with this error in judgment, he was given 1,000 c.c. of physiological saline by hypodermoclysis. Within a few minutes after completion of hypodermoclysis he went into a Jacksonian type of convulsion starting in his right hand and spreading all over his body with such violent contractions that it took four people to hold him in bed. Prompt intravenous glucose and subsequent general dehydration stopped his convulsions, and he lived to complete recovery. I believe this man had a subdural hematoma over the right motor area. This hematoma did not exert pressure as long as the volume of cerebral spinal fluid was controlled, and intracranial pressure reduced. But I believe I could have brought on his convulsions at any time by simply increasing his fluid intake.

To return to the subject of spinal punctures. Although they should not be used repeatedly to control cerebral pressure, they have a very definite place in the treatment of head injuries. As an aid in diagnosis, blood found in the spinal fluid is a certain indication that there is a laceraton or lesser bleeding point communicating with the subarachnoid spaces. Do not worry much about confusing blood present in the cerebro-spinal fluid with that due to trauma from the spinal needle itself. The latter will only be present in the first few c.c. and then will rapidly diminish. But if the blood is coming from higher up, it will be uniformly mixed with the spinal fluid, with just as much blood in the last portion withdrawn as the first.

If some time has passed before spinal puncture is done, the blood cells may be partly crenated and resemble leukocytes in a microscopic examination. In that case it will be wise to do a benzidine test for blood on this specimen to help differentiate from a beginning meningitis. If a bloody spinal fluid is found, then a daily spinal puncture is indicated, not to control the volume of cerebral spinal fluid, but to remove the blood cells. The blood cells themselves are believed to be irritating to the cortex, thus producing more edema. Blood cells are also believed to be a mechanical obstruction to the absorption of cerebro-spinal fluid through the walls of the venous sinuses. These cells will not disappear in a short time if left alone, but will remain suspended in the cerebro-spinal fluid for many days. To remove them, a daily spinal puncture should be done, draining out all the spinal fluid that can be obtained with the patient in the reclining position. This may amount to from 30 to 100 c.c. per puncture, and will average 45 to 65 c.c.

We must not forget, however, that the absence of blood in the cerebro-spinal fluid does not necessarily mean that there is no cerebral hemorrhage present. To obtain blood by spinal puncture, the bleeding must be in the subarachnoid spaces. We may have enormous hemorrhage and clots subdurally and epidurally within the skull, without blood in the cerebro-spinal fluid.

If the patient is seen within a short time after receiving a severe head injury, he will invariably be in some degree of shock. This will be the usual picture of shock with rapid, shallow respirations, low diastolic blood pressure, a rapid pulse, cold extremities, and a subnormal temperature. It would seem that the "nervous theory" of shock would be the one most applicable to cases of head injury. According to this theory, shock is caused by an exhaustion or inhibition of the vasomotor mechanism. There is consider-

able evidence to prove that the nuclei for this mechanism are located in the floor of the fourth ventricle. Loss of vasomotor tone causes dilatation of all small vessels throughout the body, especially those of the splanchnic circulation. When the capillaries are dilated they hold many times their normal volume of blood, and some of the plasma leaks out into the tissue spaces, actually reducing the volume of circulating blood. As a result the pressure of the general circulation is lowered, as may be seen by the low diastolic blood pressure readings of shock. This lowered blood pressure is very detrimental to cases of head injury because it further embarrasses that all-important blood volume that we are trying to maintain in the skull.

To combat this factor we give surgical pituitrin in 1 c.c. doses to constrict the capillaries. Atropine sulphate 1/100 gr. is given for the same reason and also to prevent sweating and so reduce body heat loss. The 50 c.c. of 50 per cent glucose mentioned before given intravenously is also of great value in shock, as by osmosis it draws the blood plasma back from the tissue spaces, thus building up the volume of the circulating blood. This action is in addition to its important effect on the cerebro-spinal fluid volume.

Let me apologize for the rambling form of the first part of this paper. I have tried to explain the mechanical factors present in head injuries, the abnormal conditions of the important three volumes within the skull, and the principles of their control by medical means. In the paragraphs that follow I will try to organize these principles into their proper sequence.

The treatment is best divided into stages.

The *first stage* is that of shock. This may last only a few hours, but generally persists for twelve hours. The patient should be put to bed. Pituitrin surgical (1 c.c.) should be administered with atropine 1/100 gr. to close the capillary network and elevate the diastolic blood pressure. Fifty c.c. of a 50 per cent solution of glucose should be given intravenously to regain the blood plasma lost to the tissues, and to begin the reduction of cerebro-spinal fluid volume. The pituitrin, atropine, and glucose may be repeated every four hours during shock. The body temperature should be maintained by blankets and hot water bottles. Caffeine sodium benzoate and adrenalin may be used if the shock seems severe enough to portend death by vasomotor collapse. Spinal puncture should be performed to determine whether or not there is blood in the cerebro-spinal fluid. If blood is present, all the spinal fluid that can be obtained with the patient in the reclining position should be withdrawn. This procedure may be repeated daily as long as the fluid remains blood tinged. The average amount withdrawn will be 45 to 65 c.c. A pressure reading at this time with a spinal manometer will be of value, remembering that the normal cerebro-spinal fluid pressure varies between 60 and 120 m.m. of water. If a spinal manometer is not available, a flow of about 1 drop per second through an 18-gauge needle will indicate a normal pressure.

If there is bleeding from the middle ear or nose these organs should be packed with gauze soaked in 5 per cent mercurochrome. In such cases, urotropin may be given in 20 grain doses three times daily to help prevent a meningitis, but there is no evidence to prove that it will actually do so.

As shock lessens the patient will very often be irrational and violent. Sedatives, such as 15 grains of chloral hydrate with 30 grains of sodium bromide, or two nembutal capsules will be of great value and must be repeated often enough to keep the patient quiet. Morphine in the shock stage is most likely contraindicated bebecause of its depressant action on the already shocked vital centers, particularly that of respiration.

During shock, certain procedures are contraindicated. Do not move the patient about to take x-rays. Do not operate in shock. Do not start general body dehydration in this stage. Do not give quantities of intravenous saline to combat this type of shock, or the patient may recover from his shock just in time to succumb to the cerebral edema made possible by this fluid.

The *second stage,* following shock, is that of edema and hemorrhage. This period generally exists for about two to five days. It is some twelve hours after injury that the edema and slow hemorrhage begin to show their effect on the vital centers. The symptoms may be a lapse into periods of unconsciousness, or actual coma, a lowered diastolic pressure, and a low pulse rate. Now we should bring into play our more permanent dehydration to aid our temporary glucose osmosis therapy. Fluids should be limited to 600 c.c. per twenty-four hours. But if a daily

spinal drainage is being performed to remove blood cells, the fluid intake may be safely raised to 900 c.c. However, should spinal drainage be discontinued, do not fail to return to the 600 c.c. daily fluid intake. For further dehydration, 1.5 ounces of magnesium sulphate in 6 ounces of water is given by mouth if the patient is conscious, and need not be calculated in the fluid intake because this fluid will not be absorbed. If the patient is unconscious, 3 ounces of magnesium sulphate in 6 ounces of water may be given as a retention enema for the same purpose. Although this enema may be retained but a short time, it will be found very effective in removing fluid through the lower bowel, and may be repeated as often as necessary.

The diet should be limited to solid foods of low water content. Watery vegetables should be avoided. No salt or salty foods should be included, for they will make the patient more thirsty and the salt will tend to hold water within the tissues. Sweets of all kinds, pastries, jelly, and ice cream, along with all types of alcoholic drinks should be prohibited as they, too, will only add to the patient's thirst.

The tongue should be quite dry. This will give you a valuable sign as to the patient's water balance. However, if at any time you are in doubt about the water balance, do a spinal puncture, not for spinal drainage, but to determine the cerebro-spinal fluid pressure.

The *third stage* is that of convalescence. This generally takes place from the fifth to the fourteenth day. No patient, with a head injury severe enough to cause a definite period of unconsciousness, should be allowed out of bed before the fourteenth day. During this time, if progress is satisfactory, medication can be discontinued. Cerebro-spinal fluid pressure can be maintained by limiting fluid intake to 900 c.c. per twenty-four hours. It is important, during this time, to protect the patient from developing a suggestive hysteria or a compensation neurosis. Patients with head injuries are very susceptible to suggestion during convalescence. Relatives and friends should be advised not to speak of his injury and the possible after-effects.

The *fourth and last stage* includes the sequelæ. These most frequent after-effects are headache, vertigo, disturbance of hearing or vision, and general weakness. They are generally due to continued edema of the brain with its increased pressure, or to free blood cells in the cerebro-spinal fluid. The intracranial pressure may be controlled with a continuance of limited fluid intake. This fluid intake, as a rule, should not exceed the 900 c.c. level per twenty-four hours and should be continued for at least three months. The free blood cells, if found in the spinal fluid, should be removed by repeated spinal drainage.

In conclusion, let me repeat a few important points that I hope we will all remember. Edema and slow bleeding are the usual causes of increased intracranial pressure. Increased intracranial pressure will cause immediate embarrassment to the cerebral circulation. This blood volume must be maintained at all costs. A deficiency of oxygen and nutrition to brain cells for only a few hours will cause permanent damage. Room must be provided in the skull for edema and hemorrhage. This room is furnished best by the removal of cerebro-spinal fluid through osmosis and dehydration.

If we remember these elementary principles, common sense, good judgment, and a smattering of physiology will, in a large measure, dictate the proper therapeutic procedures.

CHRONIC SUPPURATIVE OTITIS MEDIA*

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CHRONIC suppurative otitis media is attributed to two types of pathologic change: (1) to chronic infection of bone with resulting small sequestra, granulation tissue gradually undergoing hypertrophy to form polyps and hyperplasia of mucosa with infiltration of the submucosa by round cells; or (2) to stratified squamous epithelium replacing the mucous membrane of the middle ear and forming the matrix of a mass of desquamated epithelium, degenerated leukocytes and bacterial débris known as

*From the Section on Otolaryngology and Rhinology, The Mayo Clinic, Rochester, Minnesota. Read before the meeting of the Southern Minnesota Medical Association, Albert Lea, Minnesota, August 31, 1936.

a cholesteatoma. In the first of the aforementioned types, the chronic suppuration only rarely spreads beyond the confines of the middle ear itself, but in the second type it is more likely to extend into the mastoid antrum. Tuberculosis, syphilis, and malignancy present problems outside the scope of this paper.

Hadjopoulos studied the type of infection produced by various strains of streptococci. He found that there were strains of facultative anaërobes of the type of the *Streptococcus fæcalis,* that acted as pathogens in partially or totally closed chronic foci of infection. In the mastoid cells the course of the infection produced by these organisms is usually protracted, the infection tending to produce complications of a chronic nature, with abundant granulation tissue.

Wittmaack described a diploic type of mastoid, the lining membrane of the cells of which is composed of a tall columnar type of epithelium and a very vascular, hyperplastic submucosa. He expressed the belief that this type of mastoid was produced by irritation of the embryonal mucous tissue which is the origin of the cellular system of the mastoid, and that the irritation transformed the pneumatic cellular system of the mastoid into a system of the diploic type, with hyperplastic submucosa. Wittmaack expressed the opinion that this type of mucosa has a less than normal resistance to infection and favors not only chronic suppuration but also replacement of the mucosa of the middle ear and tympanic antrum by stratified squamous epithelium.

Signs and Symptoms

The general symptoms of chronic otitis media are purulent discharge from the middle ear, more or less interference with hearing and occasionally some deleterious effect on the general health. The discharge from the ear may be extremely foul or may have little or no odor; it may vary in quantity from an amount sufficient to form only a slight crust over the attic or ear drum to a quantity sufficient to require changing the pledget of cotton, with which the ear is usually plugged, several times a day. It may be mucoid, it may be largely purulent or it may contain fragments of cholesteatoma or exfoliated bone. On examination, the source of pus usually can be identified readily as the middle ear, the pus passing through a perforation of varying size in the ear drum or through a fistulous opening which enters the canal lateral to the ear drum and communicating with the attic. The site of the perforation in the tympanic membrane has no relation to the amount of impairment of hearing, which depends on the amount of scarring about the foot plate of the stapes or exceptionally on some injury to the cochlea. Many patients who have chronic suppuration of the temporal bone suffer from anemia, lack of vigor and other symptoms of absorption of toxic substances. Of chief interest to the physician, however, are the signs and symptoms which indicate whether treatment holds out hope of cure or whether surgical operation will be necessary.

Clinical Classification

In an attempt to clarify these indications, the following classification of chronic suppurative otitis media, based on clinical grounds, is presented. It is modeled on a classification long in use at The Mayo Clinic.

Type 1.—In this type, the discharge from the ear is mucoid or purulent but the odor of decaying bone or of cholesteatoma is absent. The perforation may be large or small and the mucous membrane, when it can be seen, appears normal. The history is characteristic in that there are periods of dryness which usually are terminated by the appearance of an acute infection of the upper part of the respiratory tract. The roentgenogram of the mastoid at most gives some evidence of sclerosis.

Type 2.—In this type, in addition to the findings characteristic of type 1, there may be evidences of disease of bones of the middle ear, especially of the ossicles, with hyperplastic changes in the mucous membrane, and polyps of granulation tissue which spring from the annulus tympanicus or promontory.

Type 3.—The discharge in this type is fetid and usually profuse. There may be considerable underlying disease of the bone, evidenced by polyps of granulation tissue which spring from the tympanic attic and aditus. Examination of the attic may reveal the presence of cholesteatoma. Roentgenographic evidence of sclerosis in the mastoid is usually well marked.

Type 4.—In this type the discharge varies in quantity. It may be so slight as to constitute

a small crust over a perforation in the attic but invariably it is foul and usually it has the characteristic odor of cholesteatoma. The tympanic membrane may be intact and the discharge may come through a fistula in the superior wall of the external auditory canal, without involvement of the tympanum. The perforation may, too, be in the membrana flaccida, or central or marginal perforations of the membrana tensa may be present. Polyps composed of granulation tissue may be particularly abundant and frequently originate from the region of the round or oval window. Varying degrees of nerve deafness may be present, the labyrinthine fistula test may be positive and various degrees of malfunction of semicircular canals may be present. A roentgenogram of the mastoid process may disclose definite evidence of the presence of a cholesteatoma, although negative roentgenographic evidence is of no diagnostic value.

The Decision Whether or Not to Operate

When definite evidences of impending or present complications, such as headache, convulsions, drowsiness, choked disk, sepsis, vertigo, nausea and vomiting, and palsy of the cranial nerves are present, the necessity of immediate operation is of course obvious. However, the condition should not then be classified as chronic otitis media but it should be placed under the heading of whichever complication is present. An acute exacerbation occurring in the course of chronic otitis media also should call for immediate surgical intervention, for the amount of sclerosis that usually is present tends toward the production of complications.

In chronic suppurative otitis media of type 1 or type 2, continued suppuration usually depends on inflammation in the eustachian tube and treatment directed to this usually will give a successful result. Forcing of mild silver protein through the eustachian tube is especially useful. In these same first two types, diseased adenoids and tonsils, or a septal deflection, or disease of the paranasal sinuses may be causes of continuing infection and should receive attention. Polyps of granulation tissue should be removed with caution and cut off with a snare near their origins rather than avulsed, and further reduction should be accomplished by chemical cauterization. It is to be remembered that polypoid granulations are protective, to some degree, against infection, and that their removal exposes raw surfaces to the action of pathogenic bacteria. When they originate from the cochlea or horizontal semicircular canal, their removal may precipitate acute labyrinthitis or meningitis. If meningeal or labyrinthine signs are produced by any of these procedures, surgical intervention should be immediate. In the presence of chronic, suppurative otitis media of type 3, when cholesteatoma is present, the condition may be treated by lavage with the Hartman cannula and syringe, but watery solutions should be strictly avoided for cholesteatomas are extremely hygroscopic and acute swelling of the mass may be produced. Alcohol or acetone are the fluids of choice. If necrotic ossicles are present, their removal will facilitate recovery. After gross disease has been removed, the use of boric acid dissolved in alcohol or the Sulzberg powder, consisting of one part of potassium iodide and three parts of boric acid, usually will dissipate any remaining infection.

If there is cholesteatoma in the attic, or if granulation tissue springs from the region of the antrum, attempts at treatment should not be persisted in longer than three weeks unless there is considerable evidence of improvement. Lack of improvement is evidence of extension of disease to the tympanic antrum and identifies the condition as of type 4. With extension of the disease to the tympanic antrum, especially when cholesteatoma is present, attempts at treatment are not only futile but are dangerous to the patient. Erosion of bone and exposure of important structures are produced by expansion of the cholesteatoma, and if it lies in such a situation that it cannot be removed without better exposure, operation is indicated. As Ballance stated, "Many so-called indications for the performance of the mastoid operation in cases of chronic otorrhea clearly point to the imminence or to the actual existence of one or the other of the complications which the operation is designed to avert, and, as in appendicitis, an abscess tells the tale of an opportunity lost and of danger incurred, so such a complication likewise tells the tale of lost opportunity and needless danger. Why condemn a patient with a discharging sinus in the temporal bone to harbor his disease for months or years, when no surgeon would leave

a patient with similar disease, for example chronic empyema of the chest or a sinus in the os calcis, to the caprice of fortune?"

Some Details of Surgical Technic

The surgical approach in disease of the middle ear, attic and tympanic antrum is possible by two routes: One through the external auditory meatus and the other posterior to the attachment of the auricle, through the portion of the temporal bone which overlies the tympanic antrum, aditus and attic.

The approach through the meatus seems reasonable only in those cases in which the disease is confined to the middle ear and attic, or when the tympanic membrane is too well preserved to permit of adequate intrameatal treatment. This is especially true when remnants of the ossicles are diseased and seem to be the primary cause of the discharge. If however, the middle ear is apparently not involved and the tympanic membrane is intact even if the disease seems confined to the attic, removal of the tympanic membrane would leave exposed the medial wall of the tympanum covered by a more or less normal mucous membrane. The tendency of this membrane to secrete mucus would produce a constantly discharging ear and although this would not be serious the patient would have the impression that attempts to cure his disease had failed.

When the disease is confined to the attic and the tympanic membrane has been destroyed, operative procedures are usually unnecessary or consist at most in removing the overhanging wall of the tympanic attic. When, however, the suspicion arises that the disease has extended to involve the mastoid antrum, it would seem contrary to sound surgical judgment to attempt exposure through this narrow field. As Ballance stated, "Why try in the dark meatus to carry out a partial or complete mastoid operation when the trend of modern surgery is to bring every stage of every operation into the light?" Greater force is added to this injunction if the anatomic variations that are present in many of these chronically diseased ears are recalled. In many instances the dura of the middle fossa hangs below the level of the roof of the attic, dipping laterally, and often the sigmoid sinus is far forward, separated from the mastoid antrum by only a thin shell of bone. There are instances in which the cholesteatoma has advanced medial and posterior to the sigmoid sinus and has undermined the temporal lobe. When the meatal approach is used, the risk of operation is increased and exposure is decreased for the questionable advantage of avoiding a scar of mastoidectomy; when as a rule this scar is invisible in a few months.

The fundamental difficulty in chronic suppurative otitis media is usually in replacement of the mucous membrane which normally lines the attic, aditus and antrum of the tympanum by squamous epithelium. The inherent tendency of this epithelium is to desquamate and this results in the formation of a cholesteatoma. This cholesteatoma, confined in a virtually closed cavity, erodes the surrounding bone through pressure and thus exposes to infection the structures of the inner ear, the sigmoid sinus, the temporal lobe of the cerebrum, or the anterior aspect of the cerebellum. Therefore, the problem to be solved in surgical operation for chronic, suppurative otitis media is only rarely that of eliminating disease and is usually the mechanical one of giving free exit to the products of desquamation.

When the middle ear is not involved by disease, it would seem the part of wisdom to leave the tympanic membrane to seal off the normal mucous membrane of the middle ear from the cavity formed by the radical operation, thus being fairly certain of terminating the discharge from the ear. The modified radical mastoid operation first suggested by Bondy, and lately revived by Lillie, accomplishes this purpose excellently. In this technic, before the postauricular incision is started, the membranous meatus is cut through to the bone just lateral to the attachment of the tympanic membrane. This prevents tearing of the tympanic membrane when the membranous meatus is elevated. The overhang of the attic, the "bridge" over the aditus, and the bone over the antrum are removed as in the ordinary radical mastoidectomy, but interference with the tympanic membrane and with the structures of the middle ear is avoided. The free margin of the tympanic membrane that is left after removal of its superior and posterior attachments is folded down over the protruding heads of the malleus and incus and is packed in place. This technic also tends to reduce scarring in the middle ear and thus conserves hearing.

This is a secondary consideration, however; the most important effect is the sealing off of the mucous membrane of the middle ear. As a secondary effect it is found that these cavities epithelize relatively fast because of the outgrowth of epithelium from the free margin of the tympanic membrane.

In performing the ordinary radical mastoidectomy in those cases in which the disease has involved the middle ear, the incision in the bone is started a little forward of the position of the center of the bridge and the removal of bone is kept on the slope of the mastoid process as it passes medialward to form the posterior wall of the external auditory canal. The area of bone removed extends from the level of the tegmen antri to the tip of the mastoid process. This type of removal of bone has several advantages over the more posterior removal frequently employed. It leaves a smaller cavity which takes less time to epithelize; the facial ridge is being lowered at the same time that the mastoid cortex is being removed; if a forward lying, superficially placed sigmoid sinus is present, as is frequent in these contracted mastoids, it is less endangered. If the mastoid process is the seat of widespread disease of bone, the removal of bone must, of course, be extended. After the antrum has been exposed, the overhanging bone of the attic is removed and the bridge of bone over the aditus ad antrum, which separates the middle ear from the antrum, is chiseled away. The facial ridge is then lowered as nearly as possible to the level of the horizontal semicircular canal.

A warning of approaching danger is furnished by bleeding from a small artery in the bone as the horizontal semicircular canal is approached. Particular care should be taken to remove the triangular beak of bone that overhangs the fenestra ovalis. The middle ear is then cleaned of any remnants of tympanic membrane and necrosed ossicles that may be present, together with any cholesteatoma that may have formed in the cavity of the middle ear. The tympanic opening of the eustachian tube is identified and the pulley of the tensor tympani muscle is removed. Care is taken to curet away from the facial canal. The mucous membrane is then removed from the eustachian canal with a Yankauer curet. Some caution is used in curetting medially and superiorly, as the bone between the carotid artery and the eustachian tube sometimes presents larger or smaller dehiscences. If pneumatic cells are present above or below the eustachian orifice, they should be curetted away. The ravinian ring, to which the tympanic membrane is attached, should next be removed and the tympanic bone should be lowered until the hypotympanum can be easily inspected. The surgeon should make sure, at this time, that there are no extensions of the cavity of the middle ear underneath the facial ridge. After these procedures have been carried out, the cavity formed by the radical operation should present no crannies which do not drain freely to the exterior, and of the greatest importance in accomplishing this is to have the facial ridge well lowered. If dura has been exposed over the temporal lobe or sigmoid sinus, the edges of bone about the opening are inspected to see that sharp points or chips of bone are not pressing against the dura. The dural exposure should be made sufficiently wide to allow intradural pressure to force the dura firmly against the edges of bone, and to prevent extension of disease between the dura and the bone. If cholesteatoma has been found in the tympanic cavity and mastoid antrum, its matrix is carefully left in place, for it is the best possible skin graft and is more closely applied to the underlying bone than any graft applied by the surgeon can possibly be.

The plastic or meatal flap on the auditory canal is next fashioned from the membranous meatus. There is no single plastic operation which is best suited to all cases and the posterior flap must be cut to fit. It may be said in general, however, that the conchal opening must be made sufficiently large to allow for its contraction in healing, and that the flap should never be allowed to lie on exposed dura. Whenever possible I prefer the plastic technic of Körner, for the long flap allows covering of a part of the posterior wall of the incision in the bone and produces a sufficiently wide meatal opening.

Following the plastic operation the cavity is packed through the meatus and the postauricular wound is sutured. The packing is left in place for about five days. Following removal of the pack nothing is replaced and secretions are removed by gentle suction. Care is taken to avoid curetting granulations that may form for, in my opinion, this tends to produce excessive

growth. If the diseased tissue is completely removed, healing usually takes place without incident. The important thing is to leave behind no diseased tissue. If in doing the operation fistulous tracts are found leading to the dura of the middle fossa or of the posterior fossa, they should be followed and sufficient dura exposed to uncover their source. If a fistulous tract is found penetrating the dura, it should be enlarged and the brain abscess should be evacuated. If fistulous tracts lead around the capsule of the labyrinth they should be followed to their source, even if it is necessary to sacrifice the semicircular canal system to drain the petrous apex. If this thoroughness in operating is carried out, little trouble will be experienced in securing a well epithelized cavity and skin grafting will be found unnecessary in the majority of cases.

Conclusions

1. Cases of chronic, suppurative otitis media can be divided, on clinical grounds, into a class that will respond to medical treatment and a class that requires surgical management.

2. The surgical problem to be faced is usually that of securing free escape for epithelial débris. Occasionally disease of bone may be present and then extensive exploration may be required.

3. The matrix of the cholesteatoma should be preserved if possible.

4. Details of surgical technic are of importance in the result.

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ACUTE SURGICAL MASTOIDITIS*

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IN dealing with this subject I merely wish to bring out or recall some of the principal points in the diagnosis of acute surgical mastoiditis, which is a pathological state that precedes a surgical mastoiditis. The former may exist without ever requiring operation to obtain complete restoration of health and function.

Let us, first, clearly establish in our minds what an acute mastoiditis means. It is an acute inflammation of the mastoid antrum and cells, which most always is secondary to an acute involvement in the middle ear. Very rarely is it primary. We must, also, keep in mind the anatomical and histological structures involved. The antrum is a passage in the temporal bone connecting the middle ear cavity with the pneumatic cavities in the mastoid portion of the temporal bone. All are lined with connective tissue, which is continuous throughout.

Professor Rutten of Vienna proved, by the histological study of many temporal bones, that an acute mastoiditis is present within a few hours after the onset of an acute otitis media. In his lectures he would go so far as to say that the two conditions always existed at the same time, varying only in the degree of involvement. This is also proven by the fact that pain, tenderness, and many times swelling, over the mastoid area appear within a few hours after the development of a middle ear infection. These symptoms are found in a large number of cases; however, operative intervention is only necessary in 12 to 15 per cent.

The question now arises, when does an acute otitis media or an acute mastoiditis (I mention both because when we are thinking of one we must have the other in mind) require surgery? In other words, when does it become a surgical mastoiditis? It is not difficult to arrive at this conclusion in a typical textbook case, but often it is very difficult in the atypical type.

In a typical type the history reveals that the pain in the ear occurs during or following an attack of some disease or inflammation in the upper respiratory tract, with an otorrhea following a paracentesis or spontaneous rupture of the drum and a fever of two to five days duration.

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The discharge continues to be rather profuse and three weeks later pain reappears with an elevation in temperature, there is a sagging of the posterior-superior wall of the external canal and there is tenderness either over the mastoid tip or antral region or both. In such a case surgery appears to be obvious.

On the other hand it may not be so simple a picture. Infections of the para-auricular glands and involvements in the external auditory canal must be differentiated. To do this one must first obtain an accurate and complete history, especially in regard to the onset and general systemic reaction. The hearing test must not be neglected and the drum picture should be carefully studied, observing its position as well as the presence or absence of its details. The blood picture and x-ray are at our disposal. However, either are misleading in many cases.

The foregoing is mentioned merely to keep the surgeon on his guard. Recently, a three year old child, upon whom a mastoidectomy had been performed three months previous, was brought in for an operation on the opposite side. Examination revealed a healthy looking subject with a scar over the right mastoid. The left external ear was displaced forward by an inflammation that covered the entire mastoid. The posterior-superior wall of the external canal was noticeably edematous and the tympanic membrane was only faintly visible. The history revealed that the onset was gradual and without any apparent pain. The child had suffered intermittently during the previous six months from furunculosis of the scalp and upon close examination several boils were found located over the left side of the head. The child at no time during its life had had a discharge from either ear. The temperature was 99 degrees and the white blood count was 13,000. Because of the absence of an otic discharge it was reasonably certain that the post-auricular swelling was due to an inflammation in the superficial lymph glands, secondary to the furunculosis of the scalp. After three days of treatment directed along these lines, most of the findings about the ear had subsided. No doubt the same condition had existed on the right side and had led to an error in diagnosis and an unnecessary mastoidectomy.

We have all seen infections of the para-auricular glands and furunculosis of the posterior wall of the external canal, due especially to an aspergillus infection, which was difficult to differentiate from a surgical mastoiditis.

In an atypical case the picture at the onset may be the same as it is in the typical type. On the other hand the symptoms may be quite obscure. I have seen several cases in which the tympanic membrane had been opened several times within ten to fourteen days, each paracentesis being followed by a very moderate discharge. There was no sagging of the canal wall nor edema over the mastoid area. Tenderness was present only when firm pressure was applied to the mastoid tip and the drum had a "beefy" appearance. This appearance I consider very important because its presence has in many cases been a greater aid to me than the x-ray in arriving at a decision to operate.

In one such case the mastoidectomy was performed on the twenty-eighth day after the first symptoms of impaired hearing and a deep throbbing sensation in the ear became manifest. There was no history of previous ear trouble. Upon opening the tergmen, pus under pressure appeared and there was extensive sequestration and infection in the entire area of a pneumatic structure. The antrum was very narrow and could be entered only with a probe or a small curette. A pure culture of streptococcus hemolyticus was obtained from the field of operation.

The unusual finding in this case was the very small antrum in a pneumatic mastoid. The preoperative picture would have led one to conclude that a sclerotic or diploic structure existed. The antrum in these two anatomical types is, usually, small, the pain is deep seated, the systemic reaction is moderate or absent, the x-ray gives only limited information and the history brings out the existence of otitis media in infancy or repeated attacks during childhood.

The case mentioned was atypical both in symptoms and operative findings. In my experience fully 30 per cent have been of this obscure type.

Because of limited time for this presentation it is not possible to cite other cases and the indications which led to the final decision to operate. However, it may be well to recall to your minds several ideas or rules which have been a great aid to me. They may be applied in any case of acute otitis media to determine whether there is present or not a surgical mastoiditis.

1. Operation is seldom indicated before the tenth day after the onset of the otitis media. In

other words do not operate too soon. The term hemorrhagic mastoiditis is only a descriptive phrase of early involvement; it is not a surgical mastoiditis.

2. The appearance of the tympanic membrane is a very important guide, especially if it looks like a mass of granulation.

3. Do not conclude that a mastoidectomy is necessary because the otorrhea has existed more than six to eight weeks. The removal of a polyp from the drum membrane may stop the discharge within forty-eight hours.

4. Do not make a diagnosis of mastoiditis just because there is an edema over the mastoid area and a discharge from the external auditory canal. This may be an aspergillus infection complicated by a furuncle on the posterior wall. The discharge from an aspergillus external otitis, even though of short duration, is fetid.

5. The discharge from an acute otitis media never has an odor, except in infants under eighteen months of age, when there may be a slight odor.

6. Do not conclude that there is tenderness over the mastoid tip when the pressure is applied over its posterior surface. The pressure should be applied over the anterior surface, directed inwardly and posteriorly. Pressure applied over the posterior surface of a healthy tip will cause tenderness.

7. Do not hesitate to enlarge an existing opening in the drum if the membrane is bulging.

8. Swelling of the eyelids on the affected side indicates congestion of the venous circulation over the petrous portion of the temporal bone.[1] The bleeding caused by free incision of the drum relieves the congestion and the edema subsides in a few hours. However, if the edema persists a mastoidectomy is indicated.

9. All laboratory findings should be utilized and carefully studied, but do not be guided entirely by them.

10. A careful and complete history is necessary before making a final decision. The surgeon should never fail to realize that the operation is performed not only to eradicate the infection, but also to restore function in the ear. Failure to obtain these results is, usually, because the antrum has not been opened and the diseased, cellular structures eradicated.

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SEVERE CUTANEOUS REACTIONS TO THE BARBITURATES*

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DRUG allergy or idiosyncrasy has been defined[1] as a condition of such hypersensitiveness to a drug that an effect of an unusual yet characteristic nature is produced by a quantity of the substance which, for most individuals, lacks appreciable physiologic action. Idiosyncrasies produced by drugs are entirely different from their toxic actions resulting from overdosage. The latter are usually uniform in their manifestations, while the idiosyncrasies vary in different people and in the same person at different times. Likewise, the same allergic reactions may be produced by different medications.

The wide variation in the types of eruptions that may be produced by drugs has been well discussed in the past few years by Wise and Parkhurst,[10] Eller,[2] C. Guy Lane,[5] Unger[9] and Wise and Sulzberger.[7,8,11] The usual forms of dermatitis medicamentosa and the drugs most commonly producing them were enumerated by Wise and Sulzberger[11] as:

1. Truly edematous eruptions with erythema, vesiculation, weeping and scaling (quinine, procaine hydrochloride, ephedrine, mercurials, sometimes arsphenamines, et cetera).

2. Urticarial eruptions (belladonna, atropine, the morphine group, phenolphthalein).

3. Scaly, erythematous eruptions, purely erythematous or scarlatiniform, and morbilliform and dermatitis exfoliative-like conditions (arsenic, arsphenamine, belladonna, balsams, the heavy metals).

4. Erythema multiforme-like eruptions (phenolphthalein, antipyrine, salicylates).

*From the Division of Dermatology, University of Minnesota, H. E. Michelson, M.D., Director; and the Dermatology Service, Minneapolis General Hospital, S. E. Sweitzer, M.D., Chief. Read before the Minnesota Academy of Medicine, October 7, 1936.

5. Erythema nodosum-like eruptions (iodides, bromides).

6. Acneform, furunculoid and erysipelas-like eruptions (bromides, iodides, chlorine, oils and tars, et cetera).

7. Ulcerating and vegetating eruptions (bromides, iodides).

8. Purpuric eruptions (iodides, arsphenamines, particularly sulpharsphenamine, balsams).

9. Fixed and circumscribed, erythematous or bullous and polychromatic pigmented eruptions (phenolphthalein, antipyrine and sometimes the arsphenamines).

It is the purpose of this paper to call attention to the possible dangers and especially to severe cutaneous reactions which may follow the administration of the barbiturates. This group of drugs is, at present, the most widely used of any of the agents to produce sleep. Most physicians apparently prescribe these compounds without adequate appreciation of the potential dangers involved. While it is true that in the majority of cases even the prolonged usage of the barbiturates causes no trouble, we have observed fatal reactions following the ingestion of only a few doses of these drugs. It is worthy of note that in the preceding list, Wise and Sulzberger did not mention any of the barbiturates as possible etiologic factors in drug eruptions. In a personal communication to one of us (S.E.S.), however, Sulzberger stated that the barbiturates may cause sensitization to light, leukopenia and vascular damage. R. L. Mayer,[6] in his monograph on drug eruptions in Jadassohn's Handbuch, briefly mentioned several of the barbituric acid derivatives as causative agents in the production of dermatitis medicamentosa, and cited a few reports of deaths following the administration of nirvanol and luminal.

Since the use of these drugs is so widespread, it seems that every physician should be cognizant of the possible dangers involved, and that further warning concerning untoward reactions may be timely. The point may be reached when these drugs will be subject to the same legal supervision as are the opiates at the present time. The following cases are illustrative of the severe reactions which may follow use of the barbiturates. Cases 1, 3, and 4 represent the third type mentioned in the table of Sulzberger and Wise; i.e., eruptions which were erythematous and morbilliform at the onset and progressing to exfoliative dermatitis. There appear to be no important differences between the first and third types of drug eruptions which were mentioned in Wise and Sulzberger's classification. Many drug eruptions may be erythematous and morbilliform at their onset, passing through a weeping, eczematous stage as they become more intense and finally going on to exfoliation. Case 2 represents Wise and Sulzberger's type 4 since the eruption was not unlike severe erythema multiforme.

Reports of Cases

Case 1.—A. S., white, female, forty-eight years of age, was admitted to the Minneapolis General Hospital on July 2, 1934, complaining of redness and itching of the entire skin, of two weeks' duration. About five weeks before admission (May 24, 1934) she had consulted physicians in the out-patient department concerning obesity and was given a low caloric diet and phenobarbital (½ grain) three times daily. After taking the medication approximately two weeks, a red, itchy eruption appeared on the face and neck and within a few days spread downward over the entire body. At this time there was no coryza or sore throat, she felt well and she had not been exposed to any contagious disease. The medication had been discontinued on June 14, 1934. The patient's health, before the onset of the present condition, had been excellent, but she felt feverish following the appearance of the eruption and had two chills on the day of admission.

Examination: On admission the patient's temperature was 101.5, pulse 112, and respirations 26. She appeared acutely ill and presented a generalized, bright red, macular, scaling eruption on the back and thorax. (The condition resulted from the confluence of a generalized, macular, morbilliform eruption of a few days previously.) No portion of the skin was spared, except the palms and soles. Examination of the mouth showed that the tongue and pharyngeal and buccal mucosa was intensely inflamed, hot and dry. There was bilateral enlargement of the cervical, axillary and supraclavicular lymph nodes. The lungs were apparently normal. There was a systolic murmur over the pulmonic area.

The laboratory findings were as follows:

Urine: dark, cloudy, amber
acid
albumin, 2+
sugar—negative
occ. granular casts
few R.B.C.
many pus cells

Blood: Hgb., 112
R.B.C., 6,160,000
W.B.C., 22,900
P.M.N., 38
Lymphocytes, 42
Monocytes, 1
Eosinophiles, 17
Basophiles, 2
Wassermann—negative
Kahn—negative
Blood urea nitrogen—29 mg./%
July 7, 1934, x-ray of chest: No pathologic changes.

Course: In spite of supportive therapy the patient became progressively worse, the fever rose gradually to 103.6, and she died 12 days after hospitalization (July 11, 1934).

The post-mortem findings were summarized as follows: heart, normal; pleural cavities, normal; lungs, terminal bronchopneumonia; gallbladder, 16 cm. long by 6 cm. wide, contained twenty stones (largest 1.5 cm. in diameter), and clear fluid; spleen and gastro-intestinal tract, normal; liver, fatty metamorphosis, grossly pale and cloudy; pancreas and adrenals, normal; kidneys, normal; spleen culture, negative; skin, exfoliative dermatitis.

Case 2.—K. B., aged fifty-eight, was admitted to the Minneapolis General Hospital, November 11, 1935, complaining of the coughing of blood for three days. After study on the medical service the following diagnoses were made: coronary sclerosis, auricular fibrillation and possible infarct of the lung. On November 29, 1935, the patient was given butyl-ethyl barbituric acid (grs. 3) daily, on account of general discomfort. The drug was continued almost daily (with a few lapses) until January 22, 1936.

On January 11, 1936, the patient developed a grouped vesicular eruption over the suprapubic region, elbows and thighs. The lesions, within a few days, became dark red in color (almost hemorrhagic), suggesting a diagnosis of erythema multiforme or dermatitis herpetiformis. The throat became red and swollen, on January 25, 1936, and in spite of blood transfusions, the patient died on January 27.

The findings in the blood were as follows:

	W.B.C.	HGB.
Nov. 24	9,200	77
Dec. 3	8,400	88
18	6,100	90
31	1,100	80
Jan. 8	1,500	
26	550	
26	500	68
26	1,100	(No P.M.N.'s, all lymphocytes)
27	250	

Autopsy was refused. Final diagnoses were: dermatitis medicamentosa and agranulocytosis.

Case 3.—M. L., white, female, aged sixty-seven, had suffered with bronchitis since January, 1936. On account of general discomfort and loss of sleep, her physician had prescribed sodium pentobarbital for ten days before admission to Minneapolis General Hospital on April 1, 1936. Four days after the medication was begun, the patient began to feel drowsy and noticed a generalized, itchy, red eruption. Due to the added discomfort, more sedation was advised, and the eruption became rapidly more severe until the patient's admission to the hospital.

Except for pneumonia, four years previously, the patient's health had otherwise been good.

The laboratory findings on admission were as follows:

April 4, 1936.
Blood sugar, 95 mg./%
Urine: acid
albumin, 4+
sugar—negative
hyaline casts—numerous
granular casts—numerous
pus cells—numerous
April 2, 1936.
Blood: Hgb., 84
W.B.C., 34,250
P.M.N., 81
Lymphocytes, 5
Monocytes, 1
Eosinophiles, 13
April 4, 1936: Wassermann—negative
April 3, 1936: Roentgenogram of the lungs: Pneumonic process, right lower lobe.

Course: The patient developed anuria, increasing edema of extremities and finally pulmonary edema and died on the fifth day of hospitalization (April 5, 1936). A diagnosis of acute nephritis was made by Dr. George Fahr, on April 4, 1936. Autopsy was refused.

Case 4.—S. M., white, female, aged fifty-six, had been given phenobarbital for hypertension by a physician sometime during the summer of 1935. She remained well until about August 1, 1935, when she developed symptoms of an acute upper respiratory infection and a red, itching eruption involving the entire body. The medication was discontinued on August 10, 1935, but by this time the lesions had become confluent over the chest, abdomen and back, the eyes were swollen almost shut and the throat and mouth were hot and dry. The condition went through an extremely stormy course and the patient was confined to bed for three weeks. On several occasions a lethal outcome seemed probable. The temperature varied from 100 to 102 F. The edema of the face gradually subsided and the integument completely exfoliated during the month of August. The hair and nails were entirely lost and it was several months before the skin became normal.

Discussion

The wide variations in the clinical features of drug eruptions are well known and will not be considered in detail here. It is perhaps unnecessary to say that many odd eruptions resembling, but not quite fitting, the picture of some common dermatosis may be caused by the ingestion of drugs. The clinical features of the superficial, eczematous, type of dermatitis medicamentosa which is commonly caused by the arsphenamines and, occasionally, by quinine, the barbiturates, or other drugs, are frequently indistinguishable from those of dermatitis of external origin. Jadassohn[4] studied extensively the mechanisms of sensitization and the site of sensitivity in eczematous drug eruptions, and showed that cutaneous sensitization might take place from without or within, and that contact with the same offending sub-

stance might likewise be of external or internal origin. Many cases have been recorded which illustrate this fact, such as that of Ford's[3] in which sensitization to quinine first took place on the face from the external use of hair tonic, and the eruption recurred in exactly the same areas following dermatitis on the genitals due to quinine in a contraceptive medication with absorption and transference of the drug through the blood stream to the previously affected areas. Transfer from the genitals to the face was unlikely because the hands were free from eruption.

Wise and Sulzberger reported the case of a woman who came to the clinic for treatment of acute contact dermatitis due to "dandruff cure" containing quinine. Patch tests to 1 per cent quinine hydrochloride were strongly positive. A year later the patient ingested quinine in a laxative tablet and within three hours there was a generalized eruption. This eruption healed, but the sites which had been previously affected remained eczematous for weeks. Patch tests with quinine were again positive. Similar cases, reported frequently, serve to substantiate the belief that the route by which the excitant reaches a hypersensitive tissue is not as important as the location or the point at which hypersenitiveness exists.

In drug eruptions other than the eczematous type, contact from without fails to reproduce the eruption, presumably because the site of the sensitivity is deeper than the contact layers of the epidermis. The actual mechanism, however, may well be the same, the type of eruption being governed by the localization and type of the shock tissue, which, except for the eczematous eruptions, is unknown and possibly resides in the deeper cutis, blood vessels or nerves. The first, second and fourth cases which we observed resembled the severe eczematous type of exfoliative dermatitis and from the appearance of the eruptions could have easily been confused with extensive contact dermatitis. Unfortunately, patch tests with the drug were not done on account of the generalized involvement and the rapidly fatal outcome.

As Coca has emphasized, the symptoms of drug eruptions (including the eczematous type) and serum sickness are almost identical. The cardinal symptoms are practically the same in both (fever, edema, joint pains, local edema, incubation period, changes in blood pressure, uncommon involvement of mucosa,† recurrences, et cetera). The slight differences, when present at all, are of a quantitative nature and perhaps dependent on the differences between serum and drug antigens. The mechanisms of both are, in reality, unknown and, so far as anyone can say, the same. Hence it cannot be disputed that, if the mechanism in drug eruptions is identical with that in serum sickness, the mechanism of drug eruptions, serum disease and contact dermatitis may be the same. The differences in the clinical pictures may be assumed to be due to the difference in the type of excitant and the site and type of shock tissue. The eczematous type of drug eruption serves as the connecting link between the three conditions, since the same picture can be produced either from without or within the body by the same excitant. Passive transfers, with a few questionable exceptions, fail in drug allergy, serum disease and contact dermatitis. Patch tests succeed only in contact dermatitis and drug eruptions of the eczematous type, not necessarily because the mechanisms are different, but because the site of the sensitivity in contact dermatitis and eczematous drug eruptions is superficial rather than in the deeper structures (as is the case in most drug eruptions) where the excitant cannot reach by means of patch tests.

Just as the morphology of drug eruptions is heterogeneous, the possibilities as to the localization of hypersensitiveness are multiple (superficial epidermis, capillaries and precapillary vessels of upper cutis, deeper vessels, nerves, et cetera). In contact dermatitis, when the excitant reaches the hypersensitive epidermis, the general type of response is the same, regardless of whether it is a pollen, drug or any other irritant. Quinine, in hair lotion, for example, will cause contact dermatitis; when quinine is ingested and reaches the same sensitized area, the same type of eruption results. The excitant is the same, the eruption is the same, and the mechanism of the inflammatory response is probably the same. However, in another patient, if the ingestion of quinine produces *urticaria,* the excitant has not changed, but it is possible that the site or type of shock tissue, or the mechanism has changed. It is not logical to believe that the mechanism (that is, the type of antibody if any) is different for each

†In three of our cases there was intense involvement of the oral mucosæ.

variation in the manifestations of idiosyncrasy *to the same excitant.* It is more reasonable to assume, even though it cannot be proved, that the same excitant calls forth the same mechanism which reacts, however, at different sites in the body, just as egg white in one person will cause an asthmatic attack, in another, atopic dermatitis, due to variations in the shock tissue rather than the mechanism.

After considering the question, it seems that the differences in drug allergy, serum disease and contact dermatitis are not great and that the type of excitant (serum, drugs, et cetera), the route which it takes (external, internal), and the mechanism are secondary to the site and type of tissue which the excitant ultimately reaches.

Three of our four patients (Cases 1, 3 and 4) presented dermatoses which exemplified the erythematous, eczematous type of drug eruption which, in these instances, were severe enough to cause exfoliation, systemic symptoms and death in two of the three individuals. This type of drug eruption is identical to that most commonly produced by the arsphenamines, the features of which are well known by most physicians. That the barbiturates may produce such a condition, however, is apparently not generally recognized, judging from the common use of these drugs by those who do not have a clear concept of the possible dangers which may result. In these three cases it would have been impossible, without the aid of a careful history, to definitely determine whether the eruption was caused from within by the ingestion of arsenic, quinine or the barbiturates or from without by some external agent to which the patient's integument was hypersensitive. Of course, eruptions of this type, produced from without, are usually not accompanied by severe systemic symptoms and do not end in death.

Furthermore, it is worthy of note that in two of the patients (Cases 1 and 3) symptoms of intolerance (pruritus and a mild erythematous eruption) appeared only a short time after the patients began to take the medication (two and four days, respectively). In all probability there would have eventually been a fatal outcome in Case 4 had the drug been continued.

Although Case 2 did not exemplify the eczematous type of drug sensitivity it was illustrative of the fact that similar drugs may sensitize different sites and types of tissues. In this instance the site of sensitivity was not in the contact layers of the epidermis but in deeper structures, probably blood vessels (purpuric eruption) and bone marrow (agranulocytosis), yet the drug was similar to those in Cases 1, 3, and 4, and the outcome was fatal. Although the problems of drug sensitivity are of great theoretical interest the important point which we wish to emphasize is that physicians should be alert for possible dangerous complications arising from the use of medications which are, as a rule, innocuous.

Summary

1. Attention is called to the possible dangers attending the administration of the barbiturates.

2. Four cases (three of which were fatal) of severe cutaneous reactions to these drugs are reported.

3. The theoretical considerations of drug eruptions with reference to the mechanism of sensitivity, the localization of the shock tissue and the types of eruptions are briefly presented.

4. The resemblance of drug allergy to serum disease and of certain eczematous drug eruptions to dermatitis of external origin makes it probable that the differences between these three types of allergy (drug allergy, serum disease and contact dermatitis) are not great.

5. It is believed that the site and type of hypersensitive tissue which an excitant (drug, serum or external agent) reaches is the chief factor in the type of response to that excitant, rather than the mechanism of sensitization or the route by which the excitant reaches the tissue.

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TULAREMIA WITH LOW AGGLUTINATION TITER DISAPPEARING AFTER SERUM THERAPY

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THE discovery of tularemia helped to determine the cause of some obscure fevers. Tularemia was recognized in ground squirrels in 1911 and in humans in 1914.[6] It was first reported in Minnesota in 1926.[5] Serum therapy was first used in 1931.[2] Prior to 1934 only fourteen cases had been recorded in the two major Duluth hospitals, each of which averages about 5,500 admissions yearly. Since that time, in spite of the liberal use of agglutination tests in suspicious cases, only eight additions have been made. There appears to be an almost world-wide incidence of the disease with no outstanding areas of unusual concentration.

Tularemia is a systemic disease. The variations are due to the kinds of local and general manifestations and, in all probability, to the existence of many strains and degrees of virulence of the organism. The intensity ranges from almost asymptomatic to fatal. The four types usually described are the ulcero-glandular, oculo-glandular, glandular, and typhoidal. The pneumonic type probably belongs with the latter. From 80 to 90 per cent of reported cases fall in the first two groups, which are the most readily recognized. The great majority of all have more or less pulmonary involvement.[1] Doubtless the last two occupy a variety of files in the cross indices of our hospitals. This can be understood readily and explains the suspicion that many undescribed features of the disease exist.

For this reason a single case report is presented. It describes a febrile state in which the presumptive diagnosis may be questioned, but, in so doing, it illustrates important phenomena associated with the use of Foshay serum.

Case Report

On February 18, 1935, M. O., a white woman, aged twenty-four, first consulted me. She complained of fatigue, flushing, night sweats, soreness in the left side of the neck, a burning sensation and "caking" in the eyes, respiratory pain in the left lower ribs and loss of about six pounds in the past two months.

The fatigue had been noticed for about seven weeks and was constantly increasing. Flushing had been noted for a few weeks and night sweats for a few days. She had felt feverish in the afternoon and evening for about one week. The eye discomfort had been present for about one week. The uncomfortable breathing had been increasing about the same period of time. She was getting nervous and apprehensive.

Past medical history comprised the following events. At the age of four, she had diphtheria. In 1927 an appendectomy was performed for acute appendicitis. Later in 1927 gallbladder disease with adhesions was diagnosed, the appendectomy scar was sensitive but recovery was spontaneous. In 1934 hemorrhoidectomy was performed. A window operation in March and April, 1934, for acute right maxillary sinusitis was followed by no acute recurrence of sinus disease. About November 28, 1935, soon after handling sick rabbits, a sore appeared just inside the left nostril. This persisted as a small open ulcer for about two months and then became crusted. A few days prior to my examination she had consulted another physician, who obtained a similar history and had serological tests made which were reported as showing an agglutination to Bacterium tularense in 1:40 dilution.

Examination showed an ashen pallor and a distinctly uneasy appearance. The scalp, hair and ears were normal. The pupils were moderately dilated but reacted promptly to light. Venous engorgement was marked in the conjunctiva and moderate in the fundus oculi. The membranes of the nose were moderately congested and swollen, and just inside the left nostril was a small crusted area. This corresponded to the site of the open sore mentioned in the history. Teeth and gums were in good condition. Slightly post-nasal drainage was present in a slightly injected pharynx. There was slight cervical adenopathy. The thyroid was barely palpable.

Excursion of the thin walled chest was free and equal. Some roughness of the inspiratory sounds was present over the larger bronchi but no parenchymal disease was noted. The size, shape and position of the heart were normal. Rhythm was regular, rate 84, and blood pressure 120/80. Slight exertion or excitement produced a rapid increase in the rate.

The abdomen was negative except for operative scarring. Both deep and superficial reflexes were slightly hyperactive. No bone or joint defects were noted.

The urine was normal chemically and microscopically. Hemoglobin was 68 per cent (Sahli). White blood cells numbered 11,000 per c.mm. of blood with 67 per cent polymorphonuclears, 31 per cent lymphocytes, 1

per cent monocytes, and 1 per cent eosinophiles. Oral temperature was 99 degrees F. Blood agglutination was absent for typhoid, paratyphoid and melitensis, and present with Bacterium tularense in dilution of 1:40 (the same as a few days before).* The Mantoux test gave a one plus reaction and a flat plate of the chest showed no evidence of significant pathology as reported by Dr. Gage Clement. Dr. J. R. McNutt reported x-ray films of the sinuses essentially normal except for slight thickening of the membranes about the periphery of the left maxillary sinus.

The positive agglutination report was the only definite finding. The probable diagnosis of tularemia was made and Foshay serum incorporated in the plan of treatment. After some confusion in locating the serum, a supply was obtained directly from Dr. Foshay. This was not received until March 6, 1935, sixteen days after my first examination. During this time palliative treatment had been of no help. The weakness, sweats and headaches persisted and increased, and there were more frequent periods in which the complexion was a ghastly gray. A low grade septic temperature persisted, ranging from 98.6 to 100.4.

Dr. Foshay's instructions were followed in administering the serum. An intradermal test for sensitivity was made by raising a small wheal. In a few minutes an area of erythema appeared about the site about 3 cm. in diameter. After half an hour this became slightly elevated but no urticaria or systemic reaction appeared, so 15 c.c. of the serum were given intravenously. No untoward effects were noted nor was there any appreciable change in the patient's condition. The erythema about the site of the skin test disappeared within twenty-four hours. Two days later, on March 8, 1935, 15 c.c. of serum were again given intravenously. On the following day the patient stated that she felt quite normal except for a little weakness. The temperature was normal. A blood specimen taken then was returned with a report of agglutination to Bacterium tularense of 1:20.

Her subsequent course was interesting. On March 17, 1935, one week after the 1:20 report, a report was returned showing no agglutination in any dilution. She had felt perfectly well and had been fever-free during that week and had returned to work. On the following day, however, she felt quite ill and twelve nodular masses appeared in the trunk and lower extremities. They were deep under the skin, hard, indefinitely outlined, and about 2 cm. in diameter. At first they itched. They became painful during the night and then tended to disappear in the soft tissue but became slightly ecchymotic in five areas over the shins. Ephedrine sulphate relieved the itching. In forty-eight hours she had completely recovered. On March 23, 1935, August 19, 1935, and June 5, 1936, agglutination was absent in all dilutions.

Comment

In summary, a young woman handled sick rabbits in an area known to harbor animals with tularemia. She soon developed a small, open sore in the left nostril which persisted for several weeks. She gradually developed symptoms of a general systemic infection, and felt and appeared sicker than any physical or laboratory findings could justify—with the possible exception of 1:40 agglutination reactions to Bacterium tularense on two occasions. Symptomatic treatment of various kinds did not prevent a continued increase in all the manifestations of illness. After obtaining a positive E-E (erythematous-edematous) reaction, the serum was administered in two doses of 15 c.c. each, two days apart. On the morning following the second dose the patient felt entirely well. The agglutination titer was then 1:20 and one week later it was absent and has so remained. The patient has remained well with the exception of the serum reaction.

In coming to the diagnosis of tularemia the following points must be considered. The 1:40 agglutination so long after the exposure to infection is very low. The decline to a 1:20 level at the time of clinical recovery and the subsequent absence of agglutination are at variance with reported statistics.* While the titer in non-serum treated cases tends to diminish after the eighth week, it generally remains positive indefinitely,[4] analogous perhaps to the positive Widal after typhoid fever. Then there is the intradermal test as described. This corresponds to the so-called positive edematous-erythematous or E-E reaction of Foshay.[3] He believes it to be a bacterial specific response and a valuable diagnostic aid. Lastly comes the prompt recovery following the second administration of serum. It is conceivable that this was simply a happy foreign protein effect but personal experience discourages such a view.

The essential features of this case have been reviewed by a number of physicians, some of whom possess unusual familiarity with tularemia. The report incorporates a summary of their view as well as the apparent specific value of Foshay's serum in a case presenting an unusual problem.

The author wishes to express his gratitude to

*All agglutination tests were made by the Minnesota Department of Health.

*In a personal communication in March, 1935, Dr. Foshay stated that, although he had encountered the loss of agglutinins rather frequently after the administration of goat serum, he had not previously heard of it after horse serum, which was used here.

Dr. Lee Foshay for supplying the serum and for his generous advice.

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THE SEDIMENTATION RATE IN GENERAL PRACTICE*

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WHEN one considers that the rate of red blood cell sedimentation is a simple determination of proven diagnostic and prognostic value, it seems strange that the test has not come into more general use in the practise of general medicine. The phenomenon of red blood cell sedimentation was first noted by Galen, and again by John Hunter in 1791. Little significance was attached to the phenomenon until 1918, when Fahraeus revived interest. Since that time it has been extensively used, especially on the European continent, and in the past few years has become more popular on the North American continent in larger medical centers. One of the earlier American articles was written by Westergren, who described a new technic in 1926.

There are a number of methods used at the present time with the results expressed as sedimentation rate, sedimentation time, and sedimentation index. The simplest type of test would seem to be the sedimentation rate, using the technic as described by Westergren. .4 cubic centimeter of 3.8 per cent sodium citrate is drawn into a syringe and mixed with 1.6 c.c. of blood drawn by vena puncture. This is thoroughly mixed and placed in a graduated tube which is in an upright position. The red blood corpuscles form aggregates and settle down with a different velocity in different physiological and pathological conditions. For all practical purposes, a reading at the end of one hour is sufficient. The speed of sedimentation is probably dependent on the amount of serum globulin and fibrinogen in the blood. A normal rate is considered to be anything up to 12 mm. in one hour. During menstruation, the rate may rise to 15 mm. in one hour, and during pregnancy after the first month, the rate increases to about 50 mm. in one hour. Bannick of Rochester stresses three principles which should be kept in mind: (1) a definite increase in the rate of sedimentation of the blood always reveals the presence of disease, but, with exceptions noted, of no particular disease; (2) the converse is not true—a normal rate of sedimentation does not mean that the patient has no disease; and (3) the rapidity of sedimentation gives some measure of the intensity of the disease, and when the level has been established in a given case, it provides a measure of the progress of the disease in that particular case.

The following table is based on the opinions of a number of authors regarding the effect of various pathologic conditions on the sedimentation rate.

Increased Rate

Tuberculosis
Syphilis
Arthritis
Pneumonia, empyema
Bronchiectasis
Acute rheumatic fever
Septicemia
Acute endocarditis
Malignancy
Anemia
Certain forms of blood dyscrasia
Acute coronary occlusion
Exanthemas
Acute pelvic inflammation

Normal Rate

Acute appendicitis without abscess formation
Mild upper respiratory infections
Gastric and duodenal ulcers
Functional diseases
Focal infections
Diabetes

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Hypertension
Asthma and hay fever
Mild skin diseases
Fibroma, lipoma, and simple cysts
Chronic valvular heart disease
Cardiac decompensation

In tuberculosis the sedimentation rate is an especially valuable aid in determining activity of the process. Cutler states that the test is a more reliable index of the presence or absence of activity than the temperature, pulse rate, gain in weight, or physical signs. The following case report shows the usefulness of the procedure:

Mrs. G. G., aged forty-three, gave a history of treatment for pulmonary tuberculosis of the right apex in 1926. She was discharged at that time as arrested. In July, 1936, she complained of weakness and a tired feeling. Physical examination was negative, except for impaired resonance and decreased vocal fremitus over the right apex posteriorly, and dry râles over both apices. The sedimentation rate was 10 mm. in one hour, which would indicate no activity. This finding was substantiated by a six-foot x-ray of the chest, showing old fibrosis in both apices with no apparent activity.

According to the literature, the sedimentation rate in syphilis seems to correspond to the activity. In latent syphilis, a normal rate is found.

The rapidity of sedimentation varies considerably in different malignant conditions, depending probably upon the extent of the lesion and the amount of tissue destruction. Yet relatively small malignant lesions may give a significant increase in the rate. As a rule, benign tumors have no effect on the rate. It should be stressed that patients with anemia show an increased rate, and an accurate correction for this factor has been worked out. The following case report is significant.

Mrs. H. H., aged sixty-four, was operated in March, 1934, elsewhere for carcinoma of the uterus, at which time a panhysterectomy was performed. Her convalescence was smooth until April, 1935, when she began to complain of low back pain. In May, 1935, the sedimentation rate was 54 mm. in one hour, a moderately high reading, and soon after a definite pelvic mass could be palpated. Mrs. H. died in December, 1935, as the result of metastatic carcinoma, confirming the test.

Polak of New York City has repeatedly emphasized this test as a measure of the amount of inflammation which still exists in cases of pelvic inflammation. When the operation is elective, he does not advise operation where the rate is significantly increased.

The occurrence of an increased sedimentation rate in cases of arthritis also deserves mention. Most cases of atrophic arthritis show an increased rate, while in the hypertrophic type the rate is normal. The test has long been recognized by pediatricians as an accurate test of activity in acute rheumatic fever. Rogatz of New York concludes an article on this subject by stating that in convalescent cases of acute rheumatic fever with the subsidence of acute symptoms and signs, the temperature is first to fall to normal, followed by the pulse rate, then the disappearance of immature polymorphonuclear leukocytes from the blood. Last to reach normal is the sedimentation of the erythrocytes. To prevent a child convalescing from acute rheumatic fever from getting up too soon, this test should be the criterion of inactivity. No child should be let out of bed until the sedimentation test is absolutely normal. This precaution should help to diminish serious cardiac involvement in new cases and minimize increasing damage in children already affected. The following case history illustrates this point.

Miss M. R., aged thirty, who has had two previous attacks of acute rheumatic fever, went to bed March 22, 1936, with a typical attack. Her temperature returned to normal in two weeks and her white blood cell count was never above 12,500. In eighteen days, her sedimentation rate dropped from 85 mm. in one hour to 29 mm., which is still an increased rate. At this time she was permitted to be up and around, and her rate increased to 46 mm. She was again put to bed, and at the end of seven weeks her rate had returned to 11 mm. This time it was safe for her to resume activity.

Lesser and Goldberger of New York City have written an excellent paper on the value of this test in the diagnosis of the acute abdomen. Their observations were based upon 3,000 readings in 2,000 cases over a period of two and one-half years. They conclude that the sedimentation reading in all cases of acute appendicitis, without abscess formation, or generalized peritonitis, is uniformly normal. The sedimentation readings in all other acute abdominal conditions are consistently abnormal; further, the sedimentation readings in all extra-abdominal conditions simulating the acute abdomen are consistently abnormal.

The literature concerning the sedimentation rate in coronary occlusion is unanimous in the opinion that the rate is increased in this condition. Hoffman of St. Paul states that the degree of increase is no index of the severity of the injury. He feels that a return of the rate to normal usually means healing of the infarction, but this is by no means invariably true.

Summary

1. The sedimentation test is a simple office procedure.

2. It is a valuable instrument as an aid to clinical judgment in determining prognosis, and to a slight degree in differential diagnosis.

3. It is also an important determining factor in establishing the progress of a disease.

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SOME OBSERVATIONS ON FOCAL INFECTION*

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"NO physical examination is complete without a careful examination of the tonsils." Such was the dictum of our Professor in Otolaryngology in Medical School, and such I have found to be increasingly true in general practice.

All too often, examination of the tonsils consists only of inspection. This can be very misleading, as evidenced by the following case:

A girl of nineteen was first seen because of extreme nervousness, insomnia, and restlessness. To use her own words, she was "in hysterics most of the time" and had had a "nervous breakdown" six months ago which kept her in bed for seven weeks. Under the care of several different doctors she had been treated on different occasions with sedatives, glandular products, psychotherapy, et cetera. Her basal metabolic rate was found normal. A catheterized specimen, however, showed albumin 1+ and red blood cells on microscopic examination. Inspection of the throat showed no *apparent* tonsils. In fact she had been told she had none—a statement which you have doubtless all heard from patients before. With the pillar retractor, unusually hypertrophied anterior pillars were pulled back on each side revealing submerged tonsils from which could be expressed large amounts of caseous material. This was repeated daily with rapid and marked improvement in the nervous symptoms and kidney findings. Eventually tonsillectomy was performed and the girl made a very satisfactory recovery from both the nervous and renal complications.

In a family of eleven children, four were seen who on different occasions developed these same peculiar nervous symptoms which ultimately were traced to tonsillar poisoning. Complete relief was secured in all of them by tonsillectomy. All had the submerged innocent-looking type of tonsils hiding behind the same misleading hypertrophied anterior pillars. All the tonsils were found, when exposed with the pillar retractor, to have a large slit in the upper pole which led into a labyrinth of cryptic development. Pus and blood issued with pressure. One of the boys, aged nineteen, was typical of the group; he had been having what was called a "nervous breakdown" for two and one-half months when I first saw him. The same restlessness and insomnia were present as in the case of the girl mentioned above. He had dizziness, vague pains in the head, palpitation, and some semi-convulsive seizures described in his own words as "jerky movements that lasted about an hour." There was no loss of consciousness during these attacks and no family history of epilepsy. The conclusion drawn was that these were choreiform attacks apparently from a neurotropic strain of streptococcus. Removal of the tonsils in due course of time caused complete and permanent cessation of all of these symptoms.

The arthrotropic streptococcus in tonsils is so commonly recognized that no illustration of the

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fact need be made at this time beyond the point that a number of individuals in the fifties and sixties have been seen who developed a severe "lumbago," lame shoulder, or other acute rheumatic disturbance who were ultimately cured by tonsillectomy. I only mention this to assert my belief that *age* is no contra-indication to the operation. Nevertheless, many people in this class hesitate at tonsillectomy. In such cases, it is wise, in connection with other therapy, not to overlook certain cleaning-up-of-the-tonsil procedures which I use almost routinely as preparatory measures to tonsillectomy.

These preparatory or clean-up measures consist first and foremost of expression of the contents of the crypts with the pillar retractor. The pillar is pulled back and the tonsil squeezed firmly between the instrument and the back of the throat until all of the caseous material is expressed. This process usually everts the tonsil from its fossa, causing the crypts to gape open and the purulent material to squirt out. The squeezing motion is made behind the tonsil if possible rather than directly on it, to facilitate this. Frequently some slight bleeding will occur from the inflamed crypts. An exploratory probe passed into these crypts sometimes reveals their amazing depth and size, particularly at the upper pole. Some tonsils in older people are nothing more than a sac, the whole inside being hollowed out and containing caseous material. Frequently the openings to such crypts are stenosed so that no natural drainage occurs. In a case of this type, I have found it useful to slip a cautery point bent in the shape of a hook into the crypt and with the cautery heat turned on gently to pull out on the hook enabling it to burn its way through the tonsil and leave a gaping orifice.

The limitations of such procedures must, however, be recognized and explained to patients. The crypts rapidly fill up after being emptied. It is impossible to sterilize crypts with carbolic acid or cautery or anything else because of their extensive ramifications. The extent of the tonsil-crypt system has been studied recently at Northwestern Medical School by Simonds and his coworkers. Ramifications were found up to the fifth order and the surface area of such crypt systems was computed to be as much as 90 square inches compared to a total surface area of the pharynx of only 7 inches.

Another almost insuperable difficulty is that some tonsils contain abscesses which have no demonstrable outlet through a crypt and cannot be emptied. Fortunately for diagnostic purposes such chronic intratonsillar abscesses are never present without sufficiently marked crypt infection to indicate the need for tonsillectomy. They are frequently unsuspected, however, until the tonsil is sectioned or they are ruptured in the process of tonsillectomy.

A few points of practical value for those who do tonsillectomy should bear mention here. *Always* check up on the bleeding and clotting time in advance. If either is more than three minutes give Vitamin D and some calcium preparation in large doses for a week. This will almost invariably bring both bleeding and clotting times under three minutes and render the blood-loss at the time of operation minimal. If the tonsils contain much infected material, have the patient come in every two or three days, for expression of the material. This cleaning-up process diminishes the amount of chronic inflammation and makes bleeding less likely. This is of paramount importance where kidney or heart symptoms have been traced to the tonsils. A prolonged period of cleaning up with some local application to available crypts diminishes the toxic absorption and hastens the time when operation is safe. And in this regard, it is safe to say that very few operations require better judgment than tonsillectomy in the presence of an early glomerulonephritis, or endocarditis. Another case well illustrates this point:

A young man came in for treatment of headaches which he attributed to a minor head injury sustained in an automobile accident a short time before. Examination revealed a blood pressure of 160/100 with 1+ albumin on urinalysis due to red blood cells and occasional casts. Markedly infected tonsils were found and a history obtained of numerous severe tonsillitis attacks. I started the process of expressing the tonsils every two or three days, contemplating a tonsillectomy only after some length of time when it was certain that his condition would be right for it. This rather slow approach to the problem apparently did not satisfy him as he went elsewhere and had an immediate tonsillectomy. I saw him a month later with four plus albumin, blood, and casts which have persisted ever since. I feel certain that this premature tonsillectomy definitely caused an exacerbation in his case.

In presenting this paper I wish to stress first and foremost the importance of more careful ex-

amination of the tonsils in every routine physical examination. By this is meant at least, retraction of the anterior pillar and firm pressure behind the tonsil to express the contents of the crypts. The pillar retractor should be on the examining tray of every general practitioner for both diagnostic and therapeutic use.

So much for the tonsils, and now just an observation or two about some of the other foci. It has surprised me how little apparent toxic absorption there may be from the maxillary sinus. Several silent infected maxillary sinuses have been discovered from which copious thick pus could be irrigated, that have probably been infected for years without any ascertainable constitutional reactions.

The prostate in men and the cervix in women on the other hand can cause considerable systemic trouble. Several women have been seen with the extreme nervousness and semi-hysterical symptoms described in the above tonsil cases where cautery of a big eroded cervix resulted in cure. Two or three cauterizations at intervals of six weeks were sometimes needed to effect complete healing. For the prostate, treatment has been disappointing. Massage given about every five days for six weeks with three months rest periods has been thoroughly tried. This program seems to give the best results. Elliott treatments have not helped noticeably and intravenous antiseptics only occasionally. Gonococcus filtrate has been tried on patients with a specific history with some benefit.

There appears to be a definite relation between *focal* infections of the *teeth* and *urinary infections.* Several instances in elderly people have been seen where no local cause in the urinary tract could be found for the persistence of pyuria, but where this cleared up and remained so after removal of infected teeth. This has been noticed to be especially true where pyorrhea of rather marked degree pre-existed.

It is also surprising how often apical abscesses will be found in *young* people if routine x-rays of the teeth are taken whenever a focus of infection is suspected. The not uncommon coexistence of infected teeth and tonsils in the same patient may explain the disappointing results obtained when only one focus is removed.

In regard to removal of infected teeth, I believe the same gradual approach to the problem as in tonsillectomy should be pursued. The general condition should be built up; the time for extraction should be selected in a period of remission of the general symptoms; and only a few teeth should be removed at a time, carefully watching for any reaction after each series of extractions. Flare-ups after removal of too active teeth, as you have doubtless all observed, are serious and may so aggravate an acute process as to prove fatal or leave permanent chronic changes.

The occurrence of two or more foci in the same individual is frequently encountered in relation to the infected gallbladder or appendix. One case well illustrates this: A badly diseased gallbladder with stones and pus was removed from a middle aged woman because of repeated severe local symptoms. She continued, however, to complain of attacks of rheumatoid arthritis which she had had for some time before the cholecystectomy. Only later after definitely infected tonsils were removed was any noticeable relief secured. Such combinations are probably not uncommon because there is some evidence that chronic infected tonsils or teeth predispose to gallbladder disease.

Removal of a chronic or interval appendix for relief of focal infection symptoms is apt to be disappointing. As an illustration of this I am mentioning the following case:

> A man, forty years old, came in for treatment of a lumbago which had persisted for several months after a slight injury. X-rays of the back gave negative findings. The tonsils were badly diseased but the patient refused local tonsillectomy, largely on the grounds of fear. He gave a history of several mild abdominal attacks which had been diagnosed as appendicitis. X-ray revealed a tender cecum that was fixed and did not fill well. Strangely enough he submitted to an appendectomy willingly. Numerous adhesions about the cecum were found indicating former attacks. Following appendectomy the lumbago gradually disappeared but other joint symptoms kept appearing in increasing severity. One year later tonsillectomy was performed. A chronic intra-tonsillar abscess in the lower pole of one tonsil containing a fluid yellow pus ruptured during the operation. *Fortunately* he has since had complete cessation of all rheumatic symptoms.

I used the term "fortunately" in speaking of the patient's recovery because of the time element in this case. If an active focus of infection is allowed to operate year after year unabated, the relief from its removal obviously cannot be as great as if checked in the beginning. Permanent changes are apt to take place in distant parts

of the body that cannot be undone. Thus as in cancer another heavy responsibility is placed on the general practitioner for *early* diagnosis and treatment.

In conclusion, in choosing the title "Some Observations On Focal Infection," no attempt has been made to cover the subject of focal infection to any appreciable extent or to deal with the literature on the subject, but merely to present some random observations made in *general* practice. The main point emphasized is that in the *early* detection of focal infections and in the insistence on their proper treatment, the general practitioner can render some of the most valuable service possible to his clientele.

A SIMPLE METHOD OF TRANSFUSION IN INFANTS*

LOWELL C. BARR, M.D.
Albert Lea, Minnesota

ABOUT 1660, Lohr at Oxford University transfused blood from one dog to another by means of a goose quill. Two years later Sir Christopher Wren and Archibald transfused a debauched man with 12 ounces of sheep's blood without fatality. Defibrinated blood, introduced in 1835 by Bishop, proved dangerous and so transfusion has gone through cycles of popularity and discredit. At present, it enjoys a wide confidence as a therapeutic measure in many conditions.

The restoration of confidence is due to simplification of technic and a more accurate knowledge of methods of blood typing. Shattuck in 1899 instituted blood typing and a few months later Landsteiner published a study upon the subject. These observers noted the presence of iso-agglutinins in the serum, according to which individuals could be classified into four groups. According to the Moss classification, 10 per cent of all persons are in group 1; 40 per cent in group 2; 7 per cent in group 3; and 43 per cent in group 4.

In 1898, direct transfusion was popularized by Crile. The convenient citrate method devised by D'Agate of Montpellier, has, on account of frequent secondary reactions, been largely supplanted by the transfusion of typed, unmodified blood.

Transfusion in adults is rather a simple procedure and usually is accomplished without difficulty. The veins are large, and the patients most often coöperate. But in infants and small children the difficulty is that of inserting the needle into a vein and keeping it there.

I wish to call your attention to a method which has been used for some years at the Children's Memorial Hospital in Chicago and perfected by an ex-resident, Dr. M. L. Spivek, who has recently described it.† In this method, the vein is exposed and a thread passed about it with a blunt needle. The procedure is precise and should be attended by only a few failures.

The vein most commonly used is the saphena magna, which runs just anterior to the internal malleolus and then proximally along the inner aspect of the leg. The patient is placed on a comfortable table with the operator at the foot facing the patient. The extremity is fixed to a splint, which is extremely important to prevent motion and thus dislodging the needle.

This is done by binding the foot to a padded splint, using a board somewhat wider than the leg and reaching from just above the knee to 2 cms. below the heel. This board should be padded well and attached to the lateral aspect of the leg, causing the foot to be externally rotated when the splint is flat on the table. With one-inch adhesive tape, the foot is bound to the board. From well on the underside of the board, the tape is rolled over the heel, along the inner aspect of the foot to the toes. The tape then goes around the back of the splint, arriving at the starting point. From here it goes over the instep and then around the back of the board. A second light and not constricting band is placed around the knee, binding it to the board. If this is correctly done, the fixation of the foot is definite and positive.

If a tourniquet is applied lightly above the knee, the vein will appear just anterior to the internal malleolus. If it fails to appear, slide the finger firmly across the area where the vein is sought and usually one can feel the distended vein snap and roll back under the finger.

When the vein is found, the skin is prepared with iodine and alcohol and the site of the vein is marked definitely by injecting a little 1 per cent novocaine directly over the center of the vein and the needle prick marks the exact location.

Draping is done by using one sterile towel placed on the table under the splint and leg. The second is placed across the leg with one edge just touching the needle mark; and the third parallels the second on the other side of the needle prick.

Towel clips, one on either side of the foot, are used to fasten the towel edges to the splint. The field is then exposed by turning under the edges of the towels.

Next, with a sharp pointed knife, an incision 1 cm. long is made over the vein at right angles to it; not parallel to it. The incision must be made deep enough to reveal the subcutaneous tissue in which the vein lies buried. The tissues are then spread apart and loosened by means of a mosquito forceps inserted perpendicular to the skin and spread in parallel direction to the vein.

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*Read before the annual meeting of the Southern Minnesota Medical Association, Albert Lea, Minnesota, August 30-31, 1936.

†Spivek, M. L.: Simple method of transfusion for infants and children. Jour. Pediat., 7:199-204, (Aug.) 1935.

The vein lies directly underneath the center of the incision. It is brought up by means of a small forceps slid gently underneath the vein and is identified as a glistening ribbon on the forceps.

A strand of No. 00 catgut is grasped by the forceps and drawn beneath the vein. Its ends are brought together and caught with a small snap. This strand is used to put tension on the vein in order to facilitate the further steps. A small pair of manicure scissors is used to make a nick in the wall of the vein on its upper surface. A dot of red on the white vein indicates that the lumen has been incised.

The blunt pointed 20 gauge needle is inserted into the prepared vein with the bevel down and it should slide in gently.

The needle is held in place by means of a mosquito forceps, a hole having been drilled through its tip. This is clamped on to the needle where it enters the vein and it serves to steady the needle and also to prevent leakage.

Five or 10 c.c. of normal saline solution may be injected to be sure the needle is in the lumen of the vein.

From this point on any apparatus or system of transfusion desired may be used. When the transfusion has been completed, the vein is not tied. Pressure will control bleeding until one can place a suture to close the wound. A piece of dressing controls the oozing. The same vein can be used again, since it has not been ligated.

THE PRESENT STATUS OF VITAMIN D MILK

The Council on Foods reports that of all the common foods available, milk is most suitable as a carrier of added vitamin D. Vitamin D is concerned with the utilization of calcium and phosphorus, of which milk is an excellent source. The Council has recently made the decision that for the present milk is the only common food which will be considered for acceptance when fortified with vitamin D. The properties of vitamin D may be imparted to milk by irradiation of the milk, by proper feeding of vitamin D preparations to cows and by the direct addition to milk of either natural or manufactured vitamin D concentrates. Clinical evidence of the nutritive value of each form of vitamin D milk is necessary in order to evaluate it properly. Up to Nov. 1, 1936, the Council on Foods has reviewed the evidence and accepted the following types of vitamin D milk: 1. Irradiated fresh (pasteurized) milk (produced under the Steenbrock patent) containing 135 units of vitamin D to the quart, and irradiated evaporated milk containing the same number of units to the reconstituted quart (after dilution with an equal volume of water). 2. Fresh (pasteurized) milk containing a concentrate of cod liver oil (Vitex) with 400 units of vitamin D to the quart, and evaporated milk containing the same number of units to the reconstituted quart. 3. Fresh (pasteurized) milk containing 400 units of vitamin D to the quart, the vitamin D being prepared from ergosterol by a process of activation with low velocity electrons. 4. Evaporated milk containing a cod liver oil concentrate made by the Clo-Dee (Barthen) process and containing 400 units of vitamin D to each 14½ ounces by weight. 5. Various mixtures designed for infant feeding containing cod liver oil or other sources of vitamin D. Dried milk preparations fortified with vitamin D have also been accepted. To be acceptable to the Council on Foods, bottle caps and labels for vitamin D milks must declare unitage of the vitamin D in terms of U.S.P. units and the source of the vitamin D, unless local governmental regulations prohibit such declaration. For all milks containing a minimum of 135 units, an enhanced nutritive value, especially for growing children, may be claimed; it is also permissible to state that these milks usually will prevent clinical rickets when they are fed to normal infants in customary quantities (from 1½ ounces for each pound of body weight in early infancy and 1½ pints or more daily in later infancy). For milk with only 135 units to the quart there shall be no claim or intimation that an adequate amount of vitamin D is being supplied to infants. When the milk contains as much as 400 units to the quart the claim may be made that the amount of vitamin D is greater than that usually required for the prevention of rickets in normal infants and thus that a margin of safety is offered when customary amounts of milk are taken. The foregoing statements apply equally to evaporated milk after it has been diluted with an equal volume of water. In the advertising of vitamin D milk the implication should not be made that the Council favors the use of any vitamin D fortified milk over the prescribing of other forms of vitamin D for infants or recommends the use of vitamin D milk to the exclusion of an additional supply of the vitamin in some other form. (*J. A. M. A.*, Jan. 16, 1937, p. 206.)

CASE REPORT

HEAT HYPERPYREXIA*

Report of an Extreme Case Surviving for Eight and One-half Days

CORNELIUS A. SAFFERT, M.D.

New Ulm, Minnesota

HEAT hyperpyrexia is the most severe, dramatic, and fatal form of injury due to heat. It is also called heat prostration, thermic fever, heat stroke, or sunstroke.

Symptoms.—The premonitory symptoms consist of a feeling of oppression, occipital headache, throbbing of the temples, vertigo, nausea. Pulse and respiration are rapid. Weakness, fatigue, and colored or dim vision may be present. Thirst is intense, the mouth dry, the tongue furred. Frequency and urgency are present, with but little urine passed. Perspiration, which had previously been free, ceases. Body temperature then rises rapidly and all symptoms become exaggerated. Consciousness is lost abruptly or after a period of confusion or delirium.

Physical Findings.—Coma may be profound. The pupils are contracted and react sluggishly; the conjunctivæ are injected. Respiration is deep, labored, stertorous, and may be of the Cheyne-Stokes type. The pulse usually ranges from 160-180, is of a peculiar running quality, and may be irregular. The temperature may be from 107 to 112 degrees. The deep reflexes are much weakened or absent. The skin is dry and burning hot. Cyanosis of the lips and fingers is present; the face flushed or pale. The muscular system is flaccid but twitchings show up early, and convulsions may occur. The urine is suppressed. Albumen and acetone bodies are common. Involuntary bowel movements may occur with a peculiar foul odor. The breath has a similar odor. In fatal cases the blood shows a progressive increase in non-protein nitrogen; the pulse becomes more rapid, breathing Cheyne-Stokes in type, and pulmonary edema complicates the picture. With recovery, the temperature falls gradually, consciousness returns, moderate fever persists for a few days to two weeks.

Complications.—(a) Relapse is common, especially in continued heat waves.

(b) Cardiac dilatation, with a systolic murmur may be present for some weeks. Pulmonary congestion and bronchitis are present and pulmonary edema is terminal.

(c) The deep reflexes return gradually with convalescence. With return to consciousness, there is an excited delusional state or even delirium, which in severe cases may take several weeks to clear. Difficult articulation, especially of dentals and labials may be present. Nystagmus, squint, diplopia may occur.

Sequelæ.—Deterioration in mentality, inability to concentrate, and changes in personality may persist. Repeated severe headaches are common, especially with exposure to slightly excessive temperatures. Inability to withstand even moderate temperatures may necessitate change of climate or occupation.

Treatment.—Symptomatic treatment consists in securing the coolest surroundings practicable. Hydrotherapy should be rigorously employed. It is advantageous to use the cooling power of evaporation by stripping the patient, spraying him with tepid water from a fine spray nozzle, and evaporating the water by fans. Evaporation of 1 gram of water removes 590 calories of heat, while melting 1 gram of ice removes only 80 calories. Mortality in large series where evaporation is used is 12 per cent, compared to 33 per cent in series treated otherwise. When the temperature has dropped to 102 further refrigeration should be stopped and the patient placed in dry cool surroundings. As the temperature rises repeat the hydrotherapy. Cardiac stimulation as needed. In laboratory animals caffeine prolonged resistance to death from overheating by 40 per cent over controls. Intravenous saline or saline and glucose is indicated to replace blood volume and loss of salt. After-treatment with careful nursing and neuro-psychiatric care is important.

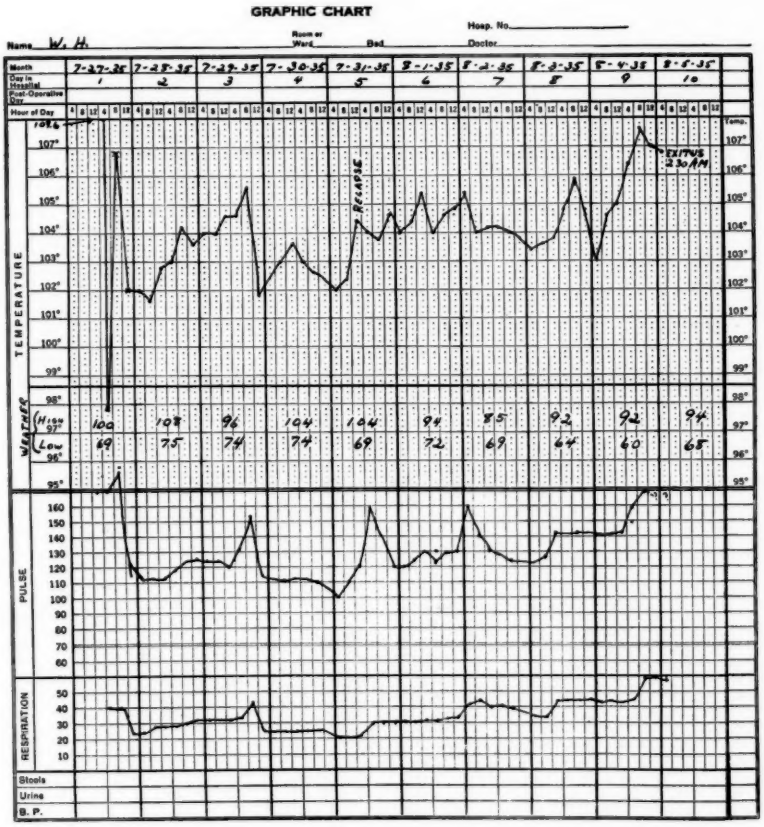
Case Report

W. H., male, aged fifty-six, weight 230, was a packer in a flour mill. On July 27, 1935, during a so-called heat wave, he was taken to his home after collapsing while at work. On arrival there he was deeply unconscious, and immediately taken to Union Hospital. Examination at 6:45 p. m.—Temperature 109.6 (R); pulse 172, thready; respiration stertorous, rate 40; B. P. 100/0; his color was ashen, skin very hot and very dry. The pupils were pinpoint in size; conjunctival reflexes absent; corneal reflex slightly present. The mucous membranes of the nose and throat were angry red, and very dry. The tongue was parched. There was a peculiar odor to the breath. The heart was very rapid and irregular but no hypertrophy nor murmurs were made out. The chest was filled with coarse and fine moist râles. There was a marked twitching of the face muscles, and occasional twitching of the muscles of the extremities. Deep reflexes were uniformly much diminished, almost absent. Abdominal reflexes were absent. Physical examination was otherwise negative.

Intensive hydrotherapy was immediately instituted, the patient being under an oxygen tent continuously. Cardiac and respiratory stimulants were given. Five hundred c.c. of 10 per cent glucose in saline were given intravenously. By 7:30 p. m. B.P. 104/60. At 7:45

*Read before the annual meeting of the Southern Minnesota Medical Association, Albert Lea, Minnesota, August 30-31, 1936.

p. m. his temperature was subnormal and external heat applied. At 8:00 p. m. his temperature was 106.8 (R), pulse 178, respiration 40. Muscular twitching of face was now marked. Hydrotherapy was again instituted. At 9:00 p. m. the B. P. was 122/76. Emesis of a foul colored, purulent sputum. Cyanosis was moderate and pulmonary edema was made out on examination. Vomiting persisted. Intravenous hypertonic glucose solution was repeated. Urine shows 2 plus albumen, otherwise negative. The red cell count and hemoglobin were nor-



brownish fluid occurred at intervals. By 11:30 p. m. the temperature had dropped to 103; pulse 114 and of good volume; respirations 24, less stertorous. The pupils were very dilated and the patient still in deep coma.

July 28, 1935.—Oxygen and other stimulants had been continued during the night. Temperature remained about 102, pulse 114, respiration 38. Retching and vomiting of foul brownish material continued at intervals. Patient still in deep coma, respirations shallow, irregular, at times Cheyne-Stokes in type. Color is pale. Temperature gradually rose to 104.2 by 8:00 p. m. with pulse and respirations slightly more rapid, cyanosis more marked. Pupils at times were pinpoint, at times dilated, reacting to light normally. Coughing occurred at intervals.

July 29, 1935.—During the night respirations were quite labored and stertorous. The morning temperature was 104, pulse 120, respiration 32; B. P. 106/86. Coma was not quite as profound but there was marked restlessness. Coughing was severe with raising of a rust mal. W. B. C. 18,050 with 88 per cent P. M. N. During the afternoon temperature rose to 105.8, pulse 142-154, respiration 42-46, shallow and stertorous. Continued extreme heat combined with the findings of pulmonary edema make the prognosis grave. Patient could not be aroused from coma. Following vigorous hydrotherapy and much stimulation, temperature by 10:00 p. m. had dropped to 101.8, pulse 116 (weaker), respiration 32, very shallow, at times of Cheyne-Stokes type. Pulse weakness responded to caffeine sodium benzoate.

July 30, 1935.—At 2:30 a. m. the patient asked for a drink. This was the first evidence of returned consciousness. He talked rationally for a moment. At 3:00 a. m. he tried to get out of bed, and wanted to return to work. He took water freely; expectorated a rust colored purulent sputum; voided normally. In the morning he was conscious but mumbled a great deal and was very difficult to understand. The tongue was very parched and black. Mucous membranes of nose and throat continued very dry and injected. Pupils reacted to light. Blood pressure was normal. Deep re-

flexes seemed to be returning. Blood chemistry showed an increase of urea and non-protein nitrogen. Chest still showed diffuse fine and coarse moist râles. Heart was regular. Temperature 103.6, pulse 112, respiration 26. By evening the temperature was about the same but there was more restlessness and some mental confusion present. Vomiting persisted and a great deal of flatus was expelled.

July 31, 1935.—Temperature this date ranged from 102-104.6, pulse 100-158, respiration 20-28. Pupils were normal in size and reacted normally. Mucous membranes of nose and throat looked somewhat better. Pulse was of good quality but respirations were still shallow at times. Chest examination still indicated pulmonary edema. Mentally he was fairly clear. The condition was improved until 9:30 a. m. when he suddenly became worse, becoming very confused and restless, picking at the bedding a great deal, mumbling incoherently and unintelligibly. He became violent at times and tried to get out of bed. He was somewhat cyanosed, the skin very hot and dry. The patient apparently had a relapse. Pulse weakness was pronounced, rate 158. Restraint had to be applied because of extreme restlessness. Patient became irrational, responding very poorly. Cough with expectoration of rust colored sputum continued, as well as vomiting. Treatment has included oxygen and carbon dioxide inhalations, caffeine and other stimulants, intravenous hypertonic glucose, intensive hydrotherapy.

August 1, 1935.—The pulse was weak at times during the night, responding, however, to caffeine. Breathing was shallow, the skin still hot and dry. The patient was restless, confused, mumbled unintelligibly, and was involuntary. The deep reflexes were less active. B. P. 154/86, temperature 104.4, pulse 128, respiration 30. Tongue and mucous membranes were parched; the cyanosis more marked. The heart was regular, the chest findings as before. Urine showed less albumin. The white blood cells numbered 14,950, 86 per cent pmn. Blood chemistery: urea 84 mgm., uric acid 13 mgm., non-protein nitrogen 201.1 mgm. per 100 c.c. By noon the temperature was 105.4, pulse 130, respiration 32, and he was extremely restless. Marked bladder distention required catheterization, the urine showing 3 plus albumin and 20-25 r.b.c. per high power field. At 5:00 p. m. venesection of 250 c.c. was done, and 25 c.c. of 50 per cent glucose administered intravenously. The mental condition remained unchanged.

August 2, 1935.—There was no response to venesection. During the night the temperature rose to 105.6, pulse 160 (irregular). Respirations were 42, labored, shallow, at times of Cheyne-Stokes in type. He rested only three hours. The scleræ were jaundiced, the pupils reacted to light. Patient talked a great deal and was rational only at intervals. Purulent sputum was still raised. The tongue was parched, the mucous membranes dry and injected. The lungs showed more râles than the day before. A marked systolic murmur was heard this date over the entire precordium. Liver and spleen were not palpable. Catheterization was again required. During the morning he again had pulse weakness with marked cyanosis with only slight response to cardiac stimulation. He was comatose at intervals. The skin again became moist, followed by profuse sweating. By evening the pulse had improved. Swallowing had become slightly easier and the patient was rational at times, but has spells of extreme restlessness. Urinalysis today showed less albumin but considerable bile. Temperature 104, pulse 124, respiration 38, throughout the night.

August 3, 1935.—Patient had a fair night. Breathing remained shallow at times and the pulse tended to be weak. While his color was better the jaundice of the scleræ remained the same. B. P. 118/76. Mucous membranes were still parched. He was mentally clear at intervals with confusion and marked restlessness at times. The pulse was of good quality. The heart still showed a bruit over all the valves. Fine and moderately coarse râles persisted throughout the chest, more marked at the right base posteriorly. Abdominal distension was less but catheterization had to be repeated. Patellar, biceps, and triceps reflexes were present on both sides. Late in the day there was increased restlessness, the pulse became weaker and somewhat irregular. There was moderate cyanosis. The blood showed moderate reduction in the retention of metabolites. There was a direct immediate reaction to the Van der Bergh test, interpreted as indicating obstruction of the bile passages due to swelling. Granular casts were present for the first time in the urine. 8:00 p. m.: temperature 105.8, pulse 142 (irregular), respiration 44.

August 4, 1935.—Temperature this date was 104.6. Cyanosis was more marked and the patient was very restless and did not recognize people. Breathing was stertorous, the heart very irregular. Catheterization had to be repeated. Twenty-five c.c. of 50 per cent glucose was given intravenously. Oxygen was administered constantly, other stimulants being continued. Restlessness was extreme and the patient had a very stormy day. Despite vigorous hydrotherapy, at 6:30 p. m. the temperature was 106.4, pulse 160, respiration 44. About the middle of the afternoon the legs began to swell, the left leg becoming very swollen and cyanosed. Delirium developed, the pulse gradually becoming weaker. Râles in the chest were increased, markedly in the dependent portions of the lungs. The neck showed moderate rigidity, the pupils being widely dilated, reacting poorly to light. At 9:00 p. m. the temperature was 107.6, respiration 56, pulse too thready to count. The entire left leg was very cyanosed and very swollen. The chest was completely filled with coarse moist râles. The patient was deeply comatose and death was imminent.

August 5, 1935.—Exitus lethalis at 2:30 a. m. on the tenth day of the illness. Permission for postmorten examination could not be obtained.

Comment—The unusually long survival of a thermic fever victim allowed a detailed clinical study which is here presented. The clinical picture can be interpreted on a basis of a severe edema of the brain, and a marked cloudy swelling of the kidneys, liver and myocardium. Terminal events were thrombosis in the lower extremities and pulmonary edema.

EDITORIAL

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A. M. A. Fellowship

EVERY physician who lays any claim to being well posted in medical matters should be conversant with the material that appears each week in the *Journal of the American Medical Association*. There is no journal that is more often referred to in medical writings and no journal that covers a wider field than our national journal. The editors of certain state medical journals rather assume that their readers are conversant with the material that appears in our national journal and try more or less to avoid duplication in material published. Whether such an assumption is justifiable is open to some question.

According to the Minnesota State Board of Medical Examiners there were 3112 licensed physicians residing in the state in 1936 and 628 additional licensed non-residents. Of these 2202 were members of our county societies and automatically members of the State Medical and the American Medical Associations. Of these state members 1412 are Fellows of the American Medical Association and receive the *Journal of the American Medical Association*. Undoubtedly a certain number of our state members subscribe to the *Journal of the American Medical Association*, but are not classed as Fellows.

When a state member subscribes to the *Journal of the American Medical Association* and pays the subscription fee of $7.00, this does not make him a Fellow. This fact is not generally understood. A member who subscribes to the national journal and who is not classified as a Fellow may apply to the national headquarters for a Fellowship. There are certain advantages in a Fellowship when it comes to attending and taking part in the scientific sessions of the American Medical Association and on moving from one state to another. If you are uncertain as to your status, consult an American Medical directory.

No part of a member's county or state dues goes to the American Medical Association. The only financial support one can give to the national organization is by subscribing to the *Journal of the American Medical Association*. If you are a county member and subscribe to the *Journal of the American Medical Association*, you should also become an American Medical Association Fellow.

Social Security Laws

THE enactment of the Social Security laws entails an enormous amount of work for federal and state departments and it is likely

to take some time before the wheels of government run smoothly. In the meantime a brief explanation of how the law affects physicians as employers may be worth while.

There are two distinct laws—one concerning the federal old age benefits and the other for federal-state unemployment compensation. Both concern the physician as employer of one or more office assistants.

The federal old age tax is entirely federal and is paid monthly to the Collector of Internal Revenue and is a 1 per cent tax on employer and employee, but is paid by the employer whether he deducts the employee's share from her salary or not. In case two or more physicians employ the same stenographer, but are not in partnership, the rather cumbersome method of making out a separate return by each physician on the quadruplicate form supplied is necessary if the physician wishes to claim exemption on his income tax for salary paid. Separate checks are sent to the Collector of Internal Revenue during the month following the month for which the salary was paid. The first installment is, therefore, payable in February for salaries paid in January, 1937. Each sheet of the return must be signed by two witnesses if the amount is less than ten dollars.

The unemployment tax, as far as the physician who employs less than eight assistants is concerned, is paid in Minnesota to the State Unemployment Compensation Division of the Industrial Commission. Returns are to be made quarterly on forms to be supplied by the State, in March. If the employees number less than eight, no tax is payable for the year 1936, but applies only to 1937. The tax is 2 per cent of the payroll in 1937 and is to be paid entirely by the employer. In the case of eight or more employees, 90 per cent of the tax is payable to the State Commission and 10 per cent to the federal internal revenue office. In case of fewer than eight employees only the 90 per cent of the 2 per cent of the payroll is to be paid to the State, the 10 per cent being omitted. In case several physicians employ the same assistants, the same rule applies as to the filing of separate returns by each physician.

Both taxes apply to one or more employees, but do not apply to domestics or agriculture laborers. Nor does the old age tax apply to employees over sixty-five years of age.

It is particularly emphasized that each month each employer must present each employee with a statement in written form of some kind as to the amount subtracted from his or her salary for the old age tax.

Catastrophes of 1937

The start of the new year is anything but propitious. It is hoped that the incidence of influenza which has reached epidemic proportions, has reached its peak and is subsiding. Each year brings a certain number of cases of influenza but not in epidemic form. It is said that the epidemic this year is not as severe as it was in 1922, 1923 and 1929, but is more severe than in 1926 and 1933. Certainly the complicating pneumonia has not made itself as conspicuous as it did in 1918, at least in Minnesota. As to the real status of the epidemic, one is forced to rely on impressions as cases are not reported.

Much discussion is heard as to the nature of the present epidemic. Whether the term "influenza" is used or the French name "la grippe" or the more popular appellation "flu" is immaterial. In our opinion the infection is the same although it varies much from year to year in incidence and severity. Little can be done to prevent its spread and the influenza bacillus is not firmly established as the etiological factor.

The importance of the influenza epidemic fades into insignificance in comparison with the flood raging in the Ohio river basin. As we go to press the high water peak has not been reached. The suffering caused the half million left homeless and the loss of property is incalculable. The public health phases of the catastrophe will be important and we are fortunate in having an organization like the Red Cross ready to assist.

These misfortunes cannot be prevented. What shall we say of the shipping and General Motors strikes? Far be it from us to pass on the justice of the present labor disputes. The suffering which the innocent are forced to share, however, is man-made and preventable.

MEDICAL ECONOMICS

Northwest Conference

EVERY physician who is interested in the social and economic relations of medicine—particularly medical society officers and committee chairmen—is urged to attend the North west Medical Conference scheduled for Sunday, February 14, at the Palmer Hotel in Chicago.

Sessions will begin at 9:30 a. m. and continue throughout the day with a luncheon for which the Iowa State Medical Society will act as host.

An important and comprehensive program by leaders in medical affairs from all over the Northwest and Midle West states has been arranged. It is printed here complete in the hope that a large representation from Minnesota will attend. Dr. W. F. Braasch, Rochester, chairman of the Committee on Medical Economics, is president of the conference, and will preside at all sessions.

Northwest Medical Conference

Palmer House
Chicago, Illinois

Sunday, February 14, 1937
8:00 a.m.—BREAKFAST—8:00 a.m.

Informal Discussion. Questions to be written and handed in—assigned to individuals for discussion.
Election of Nominating Committee

Morning Program

9:30 a.m.

President W. F. Braasch, M.D., Rochester, Minnesota, presiding

Postgraduate and Economic Education

SYMPOSIUM ON POSTGRADUATE EDUCATION:

9:30—Report of Survey—R. L. Sensenich, M.D., South Bend, Ind.
9:50—University Courses—Harold S. Diehl, M.D., Dean, University of Minnesota Medical School, Minneapolis, Minnesota.
10:00—Refresher Courses—M. H. Rees, M.D., Dean, University of Colorado School of Medicine, Denver, Colo.
10:10—Formal Local Courses—S. D. Maiden, M.D., Council Bluffs, Iowa.
10:20—Interstate Postgraduate Courses—Jas. D. McCarthy, M.D., Omaha, Nebraska.
10:30—Clinic Courses—Herman H. Riecker, M.D., Ann Arbor, University of Michigan.
10:40—Discussion led by—Ralph R. Wilson, M.D., Kansas City; M. C. Smith, Executive Secretary, Nebraska State Medical Society, Curtis, Nebraska.

SYMPOSIUM ON MEDICAL ECONOMICS:

10:55—Economic Education—E. J. Carey, M.D., Dean, Marquette University School of Medicine, Milwaukee, Wisconsin.
11:15—Economic Education of the Medical Student—Wm. J. Burns, Executive Secretary, Michigan State Medical Society, Lansing, Michigan.
11:25—Economic Education of the Doctor—E. S. Hamilton, M.D., Kankakee, Illinois.
11:35—Discussion led by—C. F. Kemper, M.D., Denver, Colorado, T. F. Thornton, M.D., Waterloo, Iowa.
11:50—Greetings from the American Medical Association—Olin West, M.D., Secretary, Chicago.
12:05—Hospital and Health Insurance—James L. Smith, M.D., Peoria, Illinois.
12:20—Discussion led by—John R. Neal, M.D., Springfield, Illinois; Carl F. Vohs, M.D., St. Louis, Missouri; T. A. Hendricks, Executive Secretary, Indiana State Medical Society, Indianapolis.

Luncheon

12:30 noon

Guests of the Iowa State Medical Society
Remarks by President W. F. Braasch
Election of Officers for 1938.

Afternoon Program

2:00 p.m.

SYMPOSIUM ON SOCIAL SECURITY ACTIVITIES:

2:00—Survey of Activities of State Governments and State Medical Societies—Chas. S. Nelson, Executive Secretary, Ohio State Medical Society, Columbus, Ohio.
2:30—Maternal and Child Welfare—Alfred W. Adson, M.D., Mayo Clinic, Rochester, Minnesota.
2:45—Public Health Services (Resettlement Administration)—A. D. McCannel, M.D., Minot, North Dakota.
3:00—Discussion led by—S. E. Gavin, Fond du Lac, Wisconsin.
3:30—Venereal Disease Program—Arthur D. Gray, M.D., Topeka, Kansas.
3:45—Discussion led by—Paul A. O'Leary, M.D., Rochester, Minn.; Earl Whedon, M.D., Sheridan, Wyoming.
4:00—State Boards of Health—Frank Jirka, M.D., Director of Public Health, Springfield, Illinois.
4:15—Discussion led by—Philip Kreuscher, M.D., Chicago, Illinois; J. F. D. Cook, M.D., Langford, South Dakota.

President's Message

Each year brings new medical problems. The one that concerns us most at present is the care of the near indigent and the indigent patient. Numerous well meaning philanthropic individuals and social agencies are attempting frequently to solve these problems without conferring with medical men and women. We as practitioners of medicine have always assumed this responsibility; but with the ever increasing number of indigent patients, find the load too heavy. Therefore, local, state and federal aid is necessary. I am sure that each one of us is only too willing to contribute his share of charity, but it is also our duty to acquaint individuals, state legislators, social agencies and welfare boards of the local medical needs and assure these groups that members of the county medical societies stand ready to coöperate in order that all those who need special aid will receive adequate medical care, since we physicians know our patients and are therefore in a better position to advise about medical measures than non-professional social workers.

A. W. Adson, M.D.

Minnesota's New Deal

There is a new note in the message that was delivered to the Legislature by Minnesota's new governor, the Honorable Elmer A. Benson.

It is a note of assurance born of the apparently overwhelming acceptance—if votes are an indication—of the basic political philosophy that inspired it.

Governor Benson believes that government may properly finance any needed welfare activity for the people. In his opinion, the November election abundantly proved that the vast majority of the people of the United States and Minnesota agree with him.

Today's Question

The question now is simply: What are the needs of the people? How can government raise sufficient taxes to supply them?

The position of Organized Medicine in this new philosophy of government is not reactionary or, in any real sense, opposed.

Physicians, in their organization, are not concerned with philosophies of government. They are concerned solely with the maintenance of high standards for the medical care of the sick and with the protection against exploitation of any sort for those who provide that care.

All Will Agree

Interference between doctor and patient; interference with the freedom of the doctor to prescribe according to the needs of the patient; interference with the maintenance of professional standards and with post-graduate education of the doctor: All these are definite concrete evils which all physicians will oppose heartily. But these are evils that all thoughtful persons will see and oppose, also, regardless of their "rightism" or their "leftism" in America.

These identical evils have been observed in most of the European countries where government paid-for-welfare aids have been extended, as a compulsory measure, to large numbers of people. Where any similar changes that may lay a basis for such undesirable conditions in America may be proposed, Organized Medicine may be expected to oppose them vigorously.

No such sweeping changes are proposed in the governor's message, however, nor is there any rumor abroad that such changes are contemplated on Capitol hill.

Doctors Must Help

Governor Benson's New Deal message for Minnesota touches upon many matters, however, that are closely related to medicine. The entire message should be studied by physicians. Physicians must be ready to step in and assist, wholeheartedly doing their share to carry out the intentions of the governor and the Legislature and watching carefully that all provisions related in any way to care of the sick or to protection of health shall be consistent with the high standards of the profession and with the policies of the profession in Minnesota.

"We have undergone a great transformation in our attitude toward government and the things we expect government to do for us," said the governor significantly in this message. "Government is no longer a mere huge policeman, protector of the rights of private property; it is now the guarantor of social and economic justice and security for all the people."

Co-operatives

Among the matters of especial interest to doctors in the message were the following:

1. Assistance to coöperatives of every description.

"You should be liberal," said the governor, "in the appropriations to the department of agriculture in its

activities relative to coöperatives. I recommend a change in the coöperative law in order to permit cooperatives a greater latitude in the purchase of stock in other corporations. . . ."

2. Legislation to make Workmen's Compensation compulsory on all employers.

"The files of the state Industrial Commission reveal that there are about 320 awards of compensation totalling approximately $360,000 which have not been paid to injured workmen or their widows and dependents because the employers carry no insurance and were themselves financially irresponsible. . . . You should increase compensation benefits generally. . . . Consideration should be given by you to the proposal for a state fund for workmen's compensation."

3. Liberalization of the garnishment law.

"I recommend liberalization of the present garnishment law so that all the wage earner possesses is not covered in the garnishment proceedings."

For Health

4. Health and recreational services.

"I urge consideration of expansion of educational facilities for unfortunate people suffering any kind of handicap. I urge . . . appropriations in the general field of health and recreational services sufficient to enable the department of education adequately to carry on a real program."

5. Department of Public Welfare.

"All public services, with the exception of supervision of public institutions under the State Board of Control, should be centered in a new department of public welfare. This would include administration of public aids under the social security act such as old-age assistance, assistance to dependent children, aid to the blind, as well as general home relief. It would call for the coöperation of the various counties through establishment of county welfare boards which would administer social assistance and treatment under the supervision of the state department."

Social Security Grants

6. Legislation that will enable Minnesota to quality for all federal grants under the Social Security Act.

"Passage by congress of the federal social security act meant that the government recognized its obligation as guarantor of the social and economic security of the people. The standards which the law sets are still far from desirable but at least it is a beginning.

"Bills must be introduced at this session whose passage will qualify the state to receive federal grants pertaining to aid to dependent children and aid to the blind."

7. Old-Age Assistance.

"The special session called by Governor Olson passed the old-age assistance act to enable the state to qualify for federal assistance. I recommend more liberal allowance so that our old folks can enjoy at least a measure of comfort." *(Medical, dental, hospital and nursing care in excess of the stipulated assistance was allowed in the first bill.—The Editors.)*

8. State Veterans' Bureau.

"I recommend that you establish a veterans' bureau which would have the function of supervision of the soldiers' home, organizing general home relief and assistance to veterans and their families."

The President's Department of Social Welfare

The Board of Trustees of the American Medical Association went on record recently as opposed to a federal department of social welfare that should include, under the control of a layman, all health activities of the federal government.

Subsequently an editorial appeared in the *Journal of the American Medical Association* urging all physicians to call the attention of legislators to this action of the board and to the desirability of maintaining the separation from and independence of health activities from general welfare work.

The president made his recommendations based upon the studies of committees of the Senate and the House on Tuesday, January 12.

New Recommendations

The recommendation called for "a department of social welfare which is to advise on social welfare problems and *also to administer federal health, education and social activities;* to conduct research in these fields; to administer federal grants, if any, for such purposes; to protect the consumer; to conduct federal aspects of federal-state programs of social security; to administer all federal eleemosynary, corrective and penal institutions and to administer probation and parole."

So ran the news report. This is the time, while the president's recommendation is taking legislative form, for members to voice their views to their representatives and senators.

In Minnesota

The House of Delegates of the Minnesota State Medical Association went on record last May as definitely opposed to any reorganization of welfare in Minnesota that should call for the merging of the State Board of Health with other agencies under a general Board of Public Welfare.

Thus far no suggestion of any such undesirable change has been made in Minnesota.

There are a number of different proposals for the necessary reorganization of relief and welfare work now being considered on Capitol hill. Governor Benson, in his message, proposed a State Board of Public Welfare (see above), with counterparts in each county to administer county welfare work. Neither the governor's nor alternative proposals provide for the inclusion of the State Board of Health in the reorganization.

Distinguished Record

Minnesota's Board of Health has proved itself, over a long period, a progressive, responsible, effective agency. Its accomplishments have been distinguished and its administration has offered no cause for the reformers to offer suggestions for change.

Reorganization of many other departments of public welfare in Minnesota are undoubtedly needed to avoid duplication and waste.

Wanted: Survey Volunteers

A letter, sent recently to virtually the entire membership of the Minnesota State Medical Association, constitutes the first step in preparation for the Council's study of the costs of medical practice in Minnesota.

This study was ordered by the Council at its December meeting at the recommendation of a committee comprised of Dr. W. W. Will, chairman, Dr. A. W. Adson and Dr. E. A. Meyerding. The committee was enlarged to include Dr. H. S. Diehl, dean of Medical Sciences, and Dr. T. H. Sweetser, chairman of the State Health Relations Committee, for conduct of the study.

Object: To secure reliable figures over a period of at least a year, based upon actual bookkeeping, to show beyond a doubt just the cost of medical practice.

Importance Grows

The importance of securing such figures grows with each move toward reorganization of medical practice for the indigent as well as for other groups.

The letter which clearly outlines the Council's purpose is printed below.

It is hoped that a representative group of physicians from every part of the state will volunteer for this work.

January 20, 1937

To: Members of the Minnesota State Medical Association

Dear Doctor:

Changes in our system of medical practice are much discussed these days.

Medical care for the indigent is being reorganized and health insurance, as a fourth factor in the administration's social security program, threatens.

In these circumstances, it is of the utmost importance to physicians to know definitely just how much it costs them to practice medicine—the cost of a medical education and of adequate post-graduate education, of office overhead, transportation, expert assistance.

Tendency to Reduce

The tendency on the part of official agencies, welfare workers, etc., is naturally to reduce fees to the lowest possible figure.

The doctor, for his own protection, for the maintenance of professional standards and for the welfare of the public, must maintain fees at a level that will permit him to maintain his standards of practice, to continue his education and to maintain himself and his family with reasonable security and in a proper standing in the community.

Exact Figures Needed

To protect medical fees, it will be absolutely necessary to have exact figures on costs—figures which are now lacking in spite of all the voluminous studies of medical care that have been piled up in the last few years.

The Council wishes to secure such figures for Minnesota and these figures cannot be secured through a brief "survey," however well organized.

They must be compiled over a long period of time, a year preferably, and by persons who have a sincere and honest interest in the work.

Appeal to Members

We are accordingly making an appeal to the membership for volunteers to do this work. We are asking for representative members in all parts of the state to volunteer to keep a careful record of all the costs of their practice for the period of a year to this end. It may require changes in bookkeeping and considerable sacrifice of time and effort on the part of the volunteers.

The information is greatly needed, however, and the final result, we are convinced, will be worth every effort it costs for the protection of our profession and the practice of medicine in the United States. Specific instructions and assistance will be sent promptly to any who volunteer to do this work.

It is our earnest hope and belief that many of our members will gladly undertake this work. The infor-

mation that only they can secure is needed equally in the urban and the rural districts, in the sparsely settled regions of the North and the closely settled farming country of the southern counties.

Sincerely yours,

W. W. WILL, M.D., *Chairman*
A. W. ADSON, M.D.
E. A. MEYERDING, M.D.
T. H. SWEETSER, M.D.
G. A. EARL, M.D.
H. S. DIEHL, M.D. (ex officio)
R. R. ROSELL (ex officio)

NOTE: Address all correspondence to Dr. E. A. Meyerding, 11 West Summit Avenue, Saint Paul, Minnesota.

Persistance Will Win

(Monthly Editorial by the Medico-Legal Advisory Committee)

"We are in the midst of a great revolution, passing from a period of extremely individualistic action into a period of associational activity," said former President Hoover some years ago.

We of the medical profession, if we study the social trends at all, must concur with him in this opinion. Beset by state medicine and all of its problems and the possible dire results to individualistic efforts, we must look to associational activity to gain the results we seek.

It is combined persistence that wins. Cato did not destroy Carthage by saying, "Carthage must be destroyed" once, but by saying it many times until he aroused the mighty Roman Empire to combined effort. Then Carthage was destroyed.

Cyrus W. Field made seven attempts at laying the Atlantic cable, but before he was successful, he must have spent much effort in convincing his associates of its feasibility.

Babe Ruth needed eight players to help him win games for his team, all coördinating as a machine to make his efforts count for all.

Your Medico-Legal Advisory Committee believes that if each member of our association will do his individual part, the combined whole will be successful. By a careful study of human nature, by sincerity of purpose and a genuine use of tact and honest judgment together with the writing of good records, on the part of all of us, the malpractice menace can be abated and eventually solved to a large degree.

Certainly properly regulated individualistic effort will make for the further security of the association in the state's economy.

County Officers' Conference

County officers, committees, the Council and interested members will hold their annual County Officers' Conference at the Lowry hotel Saturday, February 27.

This meeting affords the one opportunity of the year for secretaries and presidents and committee chairmen from all of the county and district medical societies to discuss mutually, the problems of medical organization. It also gives them a unique opportunity to carry back to their members reports of recent economic developments and plans for the future.

The morning program for the 1937 session is to begin with round table breakfast conferences for which officers and members from all the various councilor districts will be grouped for individual councilor district conferences. These breakfast conferences have been a satisfactory feature of a number of previous meetings and, incidentally, arrangements have been made with the hotel to have the tables round in fact, as well as in title, so that discussions in the various groups can be heard and shared by all. Matters of especial interest to officers of the individual societies wil occupy all of the morning session.

The afternoon will be devoted to addresses by various state and federal officials who are directly concerned in the social security and the relief programs of the state.

All Members Invited

Contemplated changes in the organization of welfare work in general and in the administration of the government's social program intimately involve the medical profession. Upon the readiness of physicians to work with state and county administrators to organize proper and lasting systems of medical care depends the future of medical practice in Minnesota and the United States.

All members are urged to attend this conference and participate in discussions. All expenses of one representative from each county and district medical society will be paid. All others who attend will be guests at luncheon of the state association.

More Deaths In England

It is of interest to note a recent article by Frederick L. Hoffman, LL.D., published in *The Weekly Underwriter,* in which he gives comparative vital statistics in England and in the United States. The incidence of most diseases was considerably higher, in many cases twice as high, in England as in the United States. Further, the improvement in the situation during the past decade was much greater in our country than in England. In many instances the rate increased in England while it decreased in this country. He makes the following notations:

"In the diseases thus far reviewed, it is therefore shown that with only two exceptions the rate was higher in England. The rate of mortality decrease is greater in this country than in England and it may be recalled that the original Act starting health insurance stated that it was 'an Act to provide for insurance against total loss of health and the prevention and cure of sickness, and for purposes incidental thereto.'

"As regards the prevention of sickness it is shown that of the diseases enumerated, most come under health insurance practice as a matter of routine experience, with much more favorable results shown for this country than for England and Wales."

No Better Test

"There is no better test than the comparative mortality figures of these two countries to illustrate the contrast between panel practice on the one hand and private medical practice on the other. The test is absolutely fair and reveals the superiority of health conditions in this country for a large majority of the wage earning element, a large proportion of which receives medical treatment free of charge. To impose a system of health insurance upon our wage earners would merely diminish their income and reduce their surplus which otherwise would go toward maintaining the American standard of life."

Our Next Meeting—Indications For Success

Interest in a medical meeting can fairly be measured by the promptness and eagerness with which commercial exhibitors engage and pay for exhibit space at the meeting.

The reason is not, of course, that commercial exhibitors are super-sensitive to the scientific excellence of a program; they are, however, very sensitive to attendance records and the interest displayed by doctors, nurses and others who attend medical meetings.

Each year the commercial exhibit section of the Minnesota State Medical Association's Annual Meeting has expanded materially over that of the previous year; but the increase for the 84th Annual Meeting to be held at the Saint Paul Auditorium May 3, 4 and 5, is so extraordinary that it is worth noting in these columns.

Exhibit spaces already engaged and paid for amount to the astonishing number of sixty-two. There are still several months before the meeting and there is a good prospect of selling many more.

A unique program comprising, not only distinguished scientific sections, but a day devoted exclusively to the first Northwest Industrial Medical Conference ever held under state association auspices, and another day—long series of sessions on the social and economic relations of all the professions associated in healing under the auspices of the Congress of Allied Professions. The importance of this last series of sessions cannot be overestimated in view of the rapidly changing face of our social organization. Famous representatives of the administration at Washington as well as medical men of national reputation will be present for the meeting.

REVISED CONSTITUTION

THE following Revised Constitution and By-Laws are to be voted upon in their final form at the Annual Meeting in May. If they are passed at that time, they will go into effect immediately.

ARTICLE I—NAME OF THE ASSOCIATION

The name of this organization is the MINNESOTA STATE MEDICAL ASSOCIATION.

ARTICLE II—PURPOSES OF THE ASSOCIATION

The purposes of this Association are to bring into one compact organization the entire medical profession of the State of Minnesota and to unite with similar societies of other states to form the American Medical Association; to promote the science and art of medicine; to elevate the standard of medical education; and to promote public health.

ARTICLE III—COMPONENT SOCIETIES AND COUNCILOR DISTRICTS

Section 1. The membership of this Association shall be organized into county and district medical societies. The functions of each such society and its relation to the Association shall be defined in a charter issued to it by the Association. Every charter so issued shall be subject to amendment and to revocation by the Association in such manner as may be prescribed in the By-Laws of the Association.

Sec. 2. A component county society is an aggregation of members of this Association living in one county.

Sec. 3. A component district society is an aggregation of members of this Association living in such districts as to make the organization of individual county societies inadvisable, or an amalgamation of two or more counties.

Sec. 4. The House of Delegates may provide for the organization of such councilor districts as will promote the welfare of the Association, such districts to be composed of component societies.

ARTICLE IV—COMPOSITION OF THE ASSOCIATION

This Association shall consist of active, affiliate, honorary, and associate members, who conform with the provisions for such membership as hereinafter provided in the By-Laws.

ARTICLE V—HOUSE OF DELEGATES

The House of Delegates shall be the governing body of the Association, and shall consist of the delegates elected by the component societies to represent them. The following shall have the privileges of the floor, but without the right to vote: the President, the President-Elect, the Councilors, the Secretary, the Treasurer, the Past Presidents, and the Delegates to the American Medical Association.

ARTICLE VI—COUNCIL

The Council shall be the executive body of the Association. The Council shall have the full authority and power of the House of Delegates between Annual Sessions, unless the House of Delegates shall be called into special session as provided for in the By-Laws. The Council shall consist of the Councilors and ex-officio but without the right to vote, the President, the President-Elect, the immediate past President, the Secretary, the Treasurer, and the Speaker of the House of Delegates. A majority of the Councilors shall constitute a quorum.

ARTICLE VII—ANNUAL SESSIONS AND MEETINGS

Section 1. This Association shall hold an Annual Session, during which there shall be held General Meetings, which shall be open to all registered members and guests.

Sec. 2. The general time and place for holding each Annual Session shall be fixed by the House of Delegates, provided that the exact date of the Session may be fixed by the Council.

Sec. 3. Special Meetings of either the Association or the House of Delegates may be called by the President on a two-thirds vote of the Council or upon petition by twenty delegates representing at least ten component societies.

Sec. 4. All Meetings of the Council may be called by the Chairman of the Council or upon petition by three Councilors.

ARTICLE VIII—OFFICERS

Section 1. The officers of this Association shall be a President, a President-Elect, two Vice-Presidents, a Secretary, a Treasurer, a Speaker and Vice-Speaker of the House of Delegates, and a Councilor for each Councilor District. These officers shall be elected by the House of Delegates as hereinafter provided in the By-Laws.

Sec. 2. There shall be elected at the Annual Session one who shall be known as President-Elect until the beginning of the next calendar year which is also the fiscal year of the Association, at which time he automatically becomes president, to serve as such for one year.

Sec. 3. The other officers, except the Councilors, shall be elected annually. The terms of the Councilors shall be for three years. As nearly as possible, one-third of the members of the Council shall be elected each year.

Sec. 4. The Delegates and Alternate Delegates to represent this Association at the House of Delegates of the American Medical Association shall be elected in accordance with the Constitution and By-Laws of the American Medical Association.

Sec. 5. Terms of office of the officers and committeemen, unless otherwise provided, shall be for a term of one year from January first following the date of their election.

All the officers shall serve until their successors are elected and installed.

Sec. 6. In case of vacancy in an office, unless otherwise provided for in this Constitution or By-Laws, the Council shall have the power to appoint temporarily a successor, until the House of Delegates shall meet and reëlect one, or until the next Annual Session.

ARTICLE IX—FUNDS AND EXPENSES

The Annual Dues shall be determined by the House of Delegates, but shall not exceed the sum of Fifteen Dollars ($15.00) per capita per annum except on a two-thirds vote of the Delegates present. Funds may also be raised from the Association's publications, by voluntary contributions, and in other manner approved by the House of Delegates. Funds may be appropriated by the House of Delegates to defray the expenses of the Association, for scientific and educational publications, and for such other purposes as will promote the advancement of medicine. All resolutions appropriating funds must be approved by the Council before action is taken thereon.

ARTICLE X—THE SEAL

The Association shall have a common Seal, and the House of Delegates shall have power to break, change or renew the same.

ARTICLE XI—AMENDMENTS

Section 1. The House of Delegates may amend any article of this Constitution by a two-thirds vote of the Delegates present at any Annual Session, provided that such amendment shall have been submitted to the membership in writing and published in the official Journal of the Association not less than three months before the meeting at which final action is to be taken.

Sec. 2. The House of Delegates, at any Annual Session, may instruct the Council to make any changes in the Articles of Incorporation in accordance with the law, which may appear desirable or which may be made necessary by any change or amendment to the Constitution and By-Laws of the Association.

Sec. 3. Upon adoption of this Constitution all previous Constitutions are thereby repealed.

ARTICLE XII—BY-LAWS

The authority for passing By-Laws to the Constitution of the Association shall be vested in the House of Delegates.

REVISED BY-LAWS

CHAPTER I—MEMBERSHIP

Section 1. All members in good standing of the component societies are members of this Association. A component society, however, which is delinquent in the payment of its annual assessments or the rendering of required reports to the Secretary of the Association shall not be permitted to participate in any of the business or proceedings of the House of Delegates or of the Association during such delinquency.

Sec. 2. Membership. The membership of this Association shall comprise all members of its component societies. Any person when he becomes a member shall agree to abide by the Articles of Incorporation, the Constitution and the By-Laws of this Association, or any changes which from time to time may be made in them, providing that he has been given notice of such change. He further agrees to abide by the Constitution and By-Laws of the Association regarding admission and expulsion and the code of ethics as laid down by the American Medical Association as it now exists or may hereafter be amended. However, any member convicted of a felony is automatically removed from membership and can only become a member by reapplication as a new member.

Sec. 3. Active Members. Active members shall comprise all the active members of component societies. No person shall be eligible for election to active membership in a component society unless he shall hold the degree of doctor of medicine, issued to him by an institution of learning accredited by the American Medical Association at the time of conferring such degree, and is licensed to practice in this state.

No person shall be considered an active member until his dues and assessments for the current year have been received at the headquarters of the Association.

Sec. 4. Affiliate Members. Affiliate members shall be those members of component societies (1) who through disability are unable to engage in the active practice of medicine, or (2) who have retired from the practice of medicine but who have been active members up to the time they applied for affiliate membership; provided however, that such member in either class shall have first been declared an affiliate member of such component society at its regular meeting, such action having been approved by the Council; and provided further, that such affiliate membership shall automatically cease and revert to its previous status upon the termination of the disability or upon the resumption of active practice. Affiliate members shall not pay dues and shall not have the right to vote or hold office.

Sec. 5. Honorary Members. The House of Delegates on recommendation of the Council may elect as honorary members any doctors of medicine who are distinguished for their services or attainments in the field of medicine, public health, research, or other scientific work contributing to medicine. Honorary members shall not pay dues and shall not have the right to vote or hold office.

Sec. 6. Associate Members. The House of Delegates on recommendation of the Council may elect as associate members any persons who are distinguished for their services in the allied sciences or in the field of public health. Associate members shall not pay dues and shall not have the right to vote or hold office.

Sec. 7. Nothing in Sections 3, 4, 5, and 6 shall in any manner invalidate an active, affiliate, honorary, or associate member in good standing at the time of the adoption of this Constitution and By-Laws.

Sec. 8. Guests. Any distinguished physician not a resident of this state who is a member of his own State Association may become a guest during an Annual Session on invitation of the officers of this Association, and shall be accorded the privilege of participating in all of the scientific work for that Session.

Sec. 9. Active members shall enjoy all the rights and privileges of the Association, including their subscriptions to MINNESOTA MEDICINE. Affiliate, Honorary, and Associate members shall have all the rights and privileges of active members except those of voting and holding office. They shall be exempt from all dues and assessments, except that they shall not be entitled to copies of MINNESOTA MEDICINE without subscription.

CHAPTER II—HOUSE OF DELEGATES

Section 1. Each component society shall be entitled to send to the House of Delegates each year one delegate, or one corresponding alternate delegate, for every fifty full-paid members, and one for any fraction thereof, but each component society which has made its annual report and paid its assessments as provided in this Constitution and By-Laws, shall be entitled to one delegate.

With the approval of the Council, a component society which previously has been entitled to a delegate or delegates, upon merging with an adjoining component society may retain its right to representation in the House of Delegates provided such society maintains a membership of five or more members.

If there are no delegates or alternate delegates from component societies present at the Annual Session, the House of Delegates may elect acting delegates from among active members of such component societies present at the Session. These acting delegates shall have all the rights and privileges of regular delegates at the Session to which they are elected, but only in the absence of the regular or alternate delegates.

Sec. 2. Twenty delegates shall constitute a quorum. All meetings of the House of Delegates shall be open to members of the Association.

Sec. 3. The House of Delegates, hereinafter termed the House, shall meet on the first day of the Annual Session. It may adjourn from time to time as may be necessary to complete its business, provided that its hours shall conflict as little as possible with the General Meetings. The order of business shall be arranged as a separate section of the program.

Sec. 4. The House shall, at its second meeting at the Annual Session, elect all the officers of the Association except the President. No delegate shall be eligible to the office of President-Elect, and no person shall be elected to any office who is not in attendance upon

that Annual Session and who has not been a member of the Association for the past two years. The Speaker and Vice-Speaker of the House may but need not be elected from among the members of the House.

Sec. 5. The House shall, at its second meeting at the Annual Session elect representatives to the House of Delegates of the American Medical Association in accordance with the Constitution and By-Laws of that body, and as is hereinafter provided.

Sec. 6. The chairman of the various appointed committees may attend the regular meetings of the House but without the right to vote. They may participate in debate on their own reports, and on invitation of the House.

Sec. 7. The Speaker, to expediate proceedings, shall appoint from the House such reference committees as he deems necessary to carry out the business of the House.

Sec. 8. The House shall, upon application, provide and issue charters to county or district societies organized to conform to this Constitution and By-Laws.

Sec. 9. The House shall divide the State into Councilor Districts, specifying what counties each district shall include, and may organize in each a medical society of the Councilor District.

CHAPTER III—ELECTION OF OFFICERS

Section 1. The manner of elections shall be determined by the assembled House, and a majority of the votes cast shall be necessary to elect.

Sec. 2. The election of officers shall be the first order of business of the House after the reading of the minutes at its second meeting.

Sec. 3. Any person known to have solicited votes for or sought any office within the gift of this Association shall be ineligible for any office for two years.

CHAPTER IV—DUTIES OF OFFICERS

Section 1. The President shall preside at all meetings of the Association except at the meetings of the House. He shall be an ex-officio member of the Council and the House, but without the right to vote. He shall appoint, with the approval of the Council, all scientific committees, not otherwise provided for. He shall deliver an annual address before the General Assembly and the House and perform such other duties as are herein provided in the Constitution and By-Laws.

Sec. 2. The President-Elect shall be an ex-officio member of the Council and House, but without the right to vote.

Sec. 3. The Vice-Presidents shall assist the President in the discharge of his duties. In case of the President's death, resignation, removal, or inability to function, the First Vice-President shall officiate during the unexpired term.

Sec. 4. The Speaker shall preside at the meetings of the House and shall perform such duties as custom and parliamentary usage require. He shall appoint the reference committees of the House. He shall have the right to vote only when his vote shall be the deciding vote. He shall be an ex-officio member of the Council but without the right to vote.

Sec. 5. The Vice-Speakers shall officiate for the Speaker in the latter's absence or at his request. In case of death, resignation, or removal of the Speaker, the Vice-Speaker shall officiate during the unexpired term.

Sec. 6. The Treasurer shall give bond in such sum as the Council may require. The Council shall execute said bond with some indemnity company at the expense of the Association. The Treasurer shall be ex-officio member of the Council and the House, but without the right to vote. He shall demand and receive all funds due the Association together with bequests and donations. He shall pay money out of the treasury only on a written order of the Chairman of the Council, countersigned by the Secretary of the Association; he shall subject his accounts to such examinations as the House may order, and he shall annually render an account of his expenditures and of the state of the funds in his hands. The amount of his salary shall be fixed by the Council. The Council may at its discretion allow the Secretary a revolving fund of such moneys as it deems advisable. This money may be expended by the Secretary for such administrative purposes as he deems necessary.

Sec. 7. The Secretary shall give bond in such sum as the Council may require. The Council shall execute said bond with some indemnity company at the expense of the Association. The Secretary shall attend the General Meetings of the Association and the meetings of the House, and shall keep minutes of their respective proceedings in separate record books. He shall be ex-officio Secretary of the Council, but without the right to vote. He shall be custodian of all record books and papers belonging to the Association, except such as properly belong to the Treasurer, and shall keep accounts of and promptly turn over to the Treasurer all funds of the Association which come into his hands.

He shall provide for the registration of the members and delegates at Annual Sessions and shall act as Business Manager for the Annual Session. Under direct supervision of the Council, he shall arrange for and have charge of the scientific and technical exhibits; collect and bank such funds received in connection thereto. He shall maintain a checking account for current expenses of the exhibits throughout the year but shall turn the balance of the funds not required into the general treasury.

He shall conduct the official correspondence, notifying members of meetings, officers of their election, and committees of their appointment and duties. He shall employ such assistants as may be ordered by the Council, and shall make an annual report to the House. He shall supply each component society with the necessary blanks for making their annual reports; shall keep an account with the component societies, charging against each society its assessments, collect the same, and at once turn it over to the Treasurer. He shall prepare and issue all programs. The amount of his salary shall be fixed by the Council. The Secretary shall present to the Association on the last day of the Annual Session, a summary of the proceedings of the Council and the House.

He shall with the coöperation of the secretaries of the component societies keep a register of all the legal practitioners of the State by counties, noting on each his status in relation to his component society, and on request, shall transmit a copy of this list to the American Medical Association.

CHAPTER V—COUNCIL

Section 1. The Council shall have full authority and power of the House between Annual Sessions, unless the House shall be called into session as provided in the Constitution and By-Laws. It shall consist of the Councilors and ex-officio but without the right to vote, the President, the President-Elect, the Secretary and Treasurer of the Association, and the Speaker of the House. A majority of its members shall constitute a quorum.

Sec. 2. The Council shall serve as the Finance Committee of the Association and perform such other functions as may be prescribed in the Constitution, By-Laws, and the Articles of Incorporation.

Sec. 3. The Council shall be the board of censors of the Association. It shall consider all questions involving the rights and standing of members, whether

in relation to other members, to the component societies, or to the Association. All questions of an ethical nature brought before the House or the General Meeting shall be referred to the Council without discussion. It shall hear and decide all questions of discipline affecting the conduct of members or component societies on which an appeal is taken from the decision of an individual Councilor, and its decision in all such matters shall be final.

Sec. 4. The Council shall meet on the first day of the Annual Session and daily during the Session and at such other times as necessity may require, subject to the call of the Chairman, or on petition of three Councilors. It shall elect a chairman and a clerk, who, in the absence of the Secretary of the Association, shall keep a record of its proceedings. It shall, through its chairman, make an annual report to the House.

Sec. 5. The Council shall provide for and superintend the publication and distribution of all proceedings, transactions and memoirs of the Association, and shall have authority to approve the appointment of the editor and such assistants as the Editing and Publishing Committee deem necessary. It shall determine the salaries of all employees of the Association. All money received by the Council and its agents, resulting from the discharge of the duties assigned to them, must be paid to the Treasurer of the Association. As the Finance Committee it shall annually supervise the auditing of the Asociation and present a statement of the same in its annual report to the House, which report shall also specify the character and cost of all the publications of the Association during the year, and the amount of all other property belonging to the Association under its control, with such suggestions as it may deem necessary.

Sec. 6. The Council shall fill any vacancy not otherwise provided for which may occur during the interval between Annual Sessions of the House; the appointee shall serve until his successor has been elected and installed.

Sec. 7. The Council shall nominate and present to the House a list of nominations for Delegates to the American Medical Association to be voted upon by the House. Additional nominations may be made from the floor of the House.

Sec. 8. The Council may at its discretion: employ expert assistance in auditing the various records of the officers and committees of the Association; employ such help as it may deem necessarv to facilitate the work of the Association; and allot for expenditures such moneys as are budgeted for the Committee on Public Policy and Legislation.

Sec. 9. The Council shall discharge such duties as are provided by law.

Sec. 10. The Council shall be empowered to invest and reinvest such monies as may be available from time to time for the creation and building up of a reserve or sinking fund. A three-fourths vote of the Council shall be necessary to authorize expenditures from this fund other than for investment or reinvestment. It may at its discretion engage the services of a Trust Company to assist in the investment and reinvestment of this fund.

Sec. 11. The Council shall appoint all non-scientific committees, not otherwise provided for in the By-Laws.

Sec. 12. Each Councilor shall be organizer, peacemaker and censor for his district. He shall visit the counties in his district when necessary for the purpose of organizing component societies where none exists; for inquiring into the condition of the profession, and for improving and increasing the zeal of the county societies and their members. The necessary traveling expenses incurred by such Councilor in the line of the duties herein imposed may be allowed by the Council on a proper itemized statement, and each Councilor may receive as compensation a per diem of $10.00 while engaged in making his official visits to the counties in his district, or in attendance at duly authorized special meetings of the Council, but this shall not be construed to include his expense in attending the Annual Session of the Association.

CHAPTER VI—COMMITTEES

Section. 1. There shall be two classes of committees: scientific and non-scientific. All committees shall consist of five members unless otherwise specified in this Constitution and By-Laws. The President may at his discretion, with the approval of the Council, increase or decrease the number of members of any committee. The President and Secretary of the Association shall be ex-officio members of all committees. All committees shall make an annual report to the House.

Sec. 2. The scientific committees shall be appointed by the President with the approval of the Council and shall consist of the following:

Scientific Assembly
  a. Local Arrangements
Cancer
Diabetes
Heart
Syphilis and Social Diseases
Deafness Prevention and Amelioration
Hospitals and Medical Education
  a. Public Health Nursing
  b. Schools for Laboratory Technicians
Maternal Welfare
Military Affairs
Historical
And such other scientific committees as may be deemed necessary.

Sec. 3. The non-scientific committees shall be appointed by the Council and shall consist of the following:

Public Policy and Legislation
Interprofessional Relationship
University Relations
Public Health Education
Editing and Publishing
Medical Economics
County Contact
And such other non-scientific committees as may be deemed necessary.

Sec. 4. The duties of the scientific committees shall be as follows:

1. Committee on Scientific Assembly. This committee shall be subdivided into three sections: Section on Medicine, Section on Surgery, and Section on Specialties. The President shall appoint annually a secretary for each of the sections, which secretary shall automatically become chairman of his section the following year, thus serving a two year term, with the exception that for the year 1938, the President shall also appoint a chairman of each section for a term of one year.

The membership of the Committee on Scientific Assembly shall consist of the Chairman and Secretary of the sections on Medicine, Surgery, and Specialties, the President, the President-Elect, the Secretary of the Association, and ex-officio, the Chairman of the Committee on Local Arrangements. The President shall act as Chairman. The Secretary of the Association shall have general charge of the arrangements and shall act as Business Manager for the scientific and technical exhibits under the direct supervision of the Council. It shall be the duty of the section chairmen to preside over the meetings of their respective sections. The

secretary of the section shall preside for the chairman in the latter's absence or at his request.

This Committee shall collaborate with the Committee on Local Arrangements to the best interests of the Annual Session.

a. The Committee on Local Arrangements. With the approval of the Council, the Committee on Local Arrangements shall be appointed by the component society of the county in which the Annual Session is to be held; it shall provide suitable accommodations for the meeting places of the Association and of the House, and of their respective committees; and the Chairman of the Committee on Local Arrangements shall assist the Secretary of the Association in making local arrangements.

2. The Committee on Cancer. This committee shall consist of fifteen members, five of which shall be appointed annually for a three year period. Its function shall be to keep the profession informed as to the latest scientific knowledge on the subject of cancer and to encourage local education of the public.

3. The Committee on Diabetes. Its function shall be to encourage the extension of medical knowledge and research into causes and treatment of diabetic disease and also to coöperate with the Committee on Public Health Education in the extension of necessary knowledge of the disease.

4. The Heart Committee. Its function shall be to promote scientific interest and progress in all phases of heart disease and to extend to the public through the Committee on Public Health Education essential education in the prevention and treatment of the disease.

5. The Committee on Syphilis and Social Diseases. Its function shall be to study the problems of prevention and control of syphilis and social diseases.

6. The Committee on Deafness Prevention and Amelioration. Its function shall be to stimulate interest in the prevention and amelioration of this affliction.

7. The Committee on Hospitals and Medical Education. Its function shall be: to give information and recommendations, if indicated to the Association in matters pertaining to medical education in the State; to encourage and develop local comprehensive programs for postgraduate instruction and in coöperation with the faculty of the University to arrange for additional courses to be given at a minimum of expense and loss of time; to maintain jurisdiction over the standardization of hospitals within the State, and in coöperation with the American Medical Association which maintains bureaus of standardization to see that such standards are maintained. The chairman of this committee shall be a representative of this Association at the annual congress of Medical Education and Medical Licensure. Public Health and Hospitals of the American Medical Association.

a. The Committee on Public Health Nursing. Its function shall be to study and act in an advisory capacity in conjunction with the Minnesota Organization for Public Health Nursing.

b. The Committee on Schools for Laboratory Technicians. Its function shall be to investigate and report on all schools for laboratory technicians; to formulate standards and makes recommendations as to the qualifications of schools as a guide to members who wish to engage the services of graduates of these schools.

8. The Committee on Maternal Welfare. Its function shall be to promote medical interest and progress in maternal and child welfare; to assist, through the Committee on Public Health Education, in the education of the public.

9. The Committee on Military Affairs. Its function shall be to maintain a constant contact with all branches of the military service and to promote and assist a proper medical coöperation at all times with the army and navy.

10. The Historical Committee. Its function shall be to assemble records of the medical history of this Association.

Sec. 5. The duties of the non-scientific committees shall be as follows:

1. The Committee on Public Policy and Legislation. This Committee shall include the President and Secretary of the Association. Under the direction of the House, it shall represent the Association in securing and enforcing legislation in the interest of public health and scientific medicine. It shall keep in touch with professional and public opinion, shall endeavor to shape legislation so as to secure the best results for the whole people, and shall strive to organize professional influence so as to promote the general good of the community in local, state and national affairs.

2. The Interprofessional Relationship Committee. Its function shall be to endeavor to bring about a better understanding and coöperation between the different interprofessional groups allied with the practice of medicine and also other groups that should coöperate in the practice of medicine.

3. The Committee on University Relations. Its function shall be to act as a contact committee with the University of Minnesota and its officials. Such problems as affect the medical profession shall be referred to this committee and they shall bring it to the attention of the proper authorities.

4. The Committee on Public Health Education. This committee shall consist of an executive committee and sub-committees.

The standing sub-committees shall be:

Child Welfare
Speakers' Bureau
Editorial
Tuberculosis
Radio
Red Cross
First Aid

The chairman may appoint such other sub-committees as he deems advisable and assign to each subcommittee as many members as he deems proper, subject to the approval of the Council.

The function of this committee shall be, first to strive to develop an intelligent public viewpoint toward the medical profession and public health education by means of the press, the lecture platform, and the radio; second, to coöperate with the various agencies throughout the state whose function is the promotion of public health, and whose governing bodies are composed in whole or in part of laymen, so that from a medical standpoint these agencies shall be intelligently administered; third, to use such measures throughout the State as may be necessary to eliminate fradulent medical advertisements from the public press; fourth, to aid and encourage each component society to conduct at least one annual public health meeting.

5. The Editing and Publishing Committee. This committee shall consist of five members, one of which shall be appointed annually for a period of five years.

The Editing and Publishing Committee shall have the responsibility of editing and publishing MINNESOTA MEDICINE; the committee shall appoint an editor and business manager and a sufficient number of assistants and shall determine their compensation; this shall be subject to the approval of the Council.

The State Association shall pay the Editing and Publishing Committee the sum of two dollars per year per active member in consideration for which each active member of the Association shall receive a copy of the Journal for one year.

Associate editors may be appointed by the Editing and Publishing Committee.

A section on medical economics shall be printed each month in MINNESOTA MEDICINE under the direction of the Council. The chairman of the Committee on Medical Economics shall have charge of this section under the title of assistant editor. He shall be ex-officio member of the Editing and Publishing Committtee and shall attend the meetings of this body.

In matters of general policy pertaining to the welfare of the Association, the Editing and Publishing Committee shall defer to the requests from the Council.

6. The Committee on Medical Economics. This committee shall consist of an executive committee composed of the chairman, the chairmen of sub-committees, and the chairmen of the Committee on Public Policy and Legislation and the Committee on Public Health Education. The duties of this executive committee shall be the coördinating of the general program. Three members shall constitute a quorum.

The sub-committees of the Medical Economics Committee shall be appointed by the chairman of the Medical Economics Committee with the approval of the Council, and shall consist of the following:

a. The Editorial Committtee. It shall be responsible for the editing and compiling of the medical economics section which is published each month in MINNESOTA MEDICINE. The chairman of the Committee on Medical Economics shall be the chairman of this subcommittee.

b. The Committee on Professional Education in Medical Ethics and Social and Economic Trends. Its function shall be: (1) to promote knowledge in medical ethics and social and economic trends among medical students and the medical profession in Minnesota. It shall endeavor to secure and maintain a course on these subjects at the Medical School at the University of Minnesota.

c. The Medico-Legal Advisory Committee. Its function shall be: (1) to study the questions pertaining to insurance, especially malpractice insurance as it affects the profession; and (2) to study and advise in legal affairs that affect the profession.

d. The Committee on State Health Relations. Its function shall be: (1) to study the activities of the various State or Governmental Agencies and their relation to the practice of medicine: and (2) to coöperate with these various State and Governmental Agencies whose function is the promotion of public health in maintaining the welfare of the public.

e. The Committee on Low Income and Indigent Problems. Its function shall be to study and present methods and plans for the care of these two groups. This committee shall make its reports to the executive committee of the Medical Economics Committee. All plans concerning the medical care of the indigent, before being presented to the membership at large must be approved by the executive committee of the Medical Economics Committee and the Council or the House.

f. The Committee on Industrial Relations. Its function shall be to discuss and recommend a policy on the various industrial questions that arise and that affect the medical profession. It shall work in coöperation with the industrial organizations to improve the conditions that affect the medical profession.

g. The Committee on Contract Practice. Its function shall be to study existing forms of contract practice and to inform and advise the executive committee of the Medical Economics Committee upon these and upon all proposed plans for contract practice in Minnesota.

7. The County Contact Committees of Three. They shall consist of three physicians practicing in the county in which they live, and appointed by their local component societies. Their functions shall be to study the medical and health problems so far as they are related to the best interests of the public and coöperate with their component medical society and the Medical Economics Committee.

CHAPTER VII—COUNTY AND DISTRICT SOCIETIES

Section 1. All county and district societies now in affiliation with the Association or those which may hereafter be organized in this State, which have adopted principles of organization not in conflict with this Constitution and By-Laws, shall, on application, receive a charter from and become a component part of the Association.

Sec. 2. As rapidly as can be done after the adoption of this Constitution and By-Laws, a medical society shall be organized in every county in the State in which no component society exists, and charters shall be issued thereto.

Sec. 3. Charters shall be issued only upon approval of the House, and shall be signed by the President and Secretary of the Association. The House shall have the authority to revoke the charter of any component society whose actions are in conflict with the letter or spirit of this Constitution and By-Laws.

Sec. 4. Only one component medical society shall be chartered in any county. Where more than one county society exists, all members should be brought into one organization. In case of failure to unite, an appeal may be made to the Council, which shall decide what action shall be taken.

Sec. 5. In sparsely settled sections the House shall have authority to organize the physicians of two or more counties into societies to be designated by hyphenating the names of two or more counties so as to distinguish them from other classes of societies, and these societies, when organized and chartered, shall be entitled to all the privileges and representation provided herein for county societies, until such counties may be organized separately.

Sec. 6. Each component society shall have general direction of the affairs of the profession in its county or district, and its influence shall be constantly exerted for bettering the scientific, moral, and educational condition of the county or district; and systematic efforts shall be made by each member, and by the society as a whole, to increase the membership until it embraces every qualified physician in the county or district.

Sec. 7 Each component society shall judge of the qualifications of its own members, but, as such societies are the only portals to this Association and to the American Medical Association, ample opportunity to become a member shall be given to every physician in the county or district, who is eligible according to the provisions in this Constitution and By-Laws.

Sec. 8. Any physician who may feel aggrieved by the action of the society of his county or district in refusing him membership, or in suspending, censoring, or expelling him, shall have the right to appeal to the Council, and if he desires, to the Judicial Council of the American Medical Association. Decision of the latter shall be final.

Sec. 9. In hearing appeals the Council may admit oral or written evidence, as in its judgment will best and most fairly present the facts, but in case of every appeal, both as a Board and as individual Councilors, in district and county work, efforts at conciliation and compromise shall precede all such hearings.

Sec. 10. A physician living on or near a county line may hold his membership in the component society most convenient for him to attend, on permission of the society under whose jurisdiction he resides.

Sec. 11. At some meeting in advance of the Annual Session, each component society shall elect a delegate or delegates and an alternate or alternates to represent it in the House, in the proportion of one delegate to

each fifty members or any fraction thereof, and the secretary of each society shall send a list of such delegates to the Secretary of the Association two months before the date fixed for the Annual Session.

Sec. 12. The secretary of each component society shall keep a roster of its members and of the non-affiliated registered physicians of the county or district, in which shall be shown the full name, address, college and date of graduation, date of license to practice in this State, and such other information as may be deemed necessary. In keeping such roster the secretary shall note any changes in the personnel of the profession by death, or by removal to or from the county or district, and in making his annual report he shall be certain to account for every physician who has lived in the county or district during the year.

Sec. 13. The secretary of each component society shall forward the assessment of its members together with its roster of officers and members, list of delegates, and list of non-affiliated physicians of the county or district to the Secretary of the Association each year before December 31.

Sec. 14. Each component society which fails to pay its assessment or make the report required, on or before December thirty-first, shall be held as suspended and none of its members or delegates shall be permitted to participate in any of the business or proceedings of the Association or of the House until such requirements have been met.

Sec. 15. The annual per capita dues to the Association of the members of the component societies shall be determined by the House and shall be paid and forwarded as hereinbefore provided, being payable on or before January first of the year for which they are levied.

CHAPTER VIII—MISCELLANEOUS

Section 1. The Articles of Incorporation, the Constitution, and By-Laws of the Association shall be binding on every county and district society and every member of every such society; anything in the Articles of Incorporation, the Constitution, or the By-Laws of any such society to the contrary notwithstanding.

Sec. 2. A member of this Association must be a member of some component society and conversely a member of a component society must be a member of this Association. An action of the House or of the Council shall be binding upon its members unless otherwise provided.

Sec. 3. The Principles of Medical Ethics of the American Medical Association shall govern the conduct of members in their relations to each other and to the public.

Sec. 4. The deliberations of the Association shall be governed by parliamentary usage as contained in Robert's Rules of Order, when not in conflict with this Constitution and By-Laws.

Sec. 5. All papers read before the Association or any of the societies shall become its property. Each paper shall be deposited with the Secretary of the Association when read.

Sec. 6. The time required for delivery of any paper or address before the Association shall be left to the discretion of the Committee on Scientific Assembly.

CHAPTER IX—AMENDMENTS

These By-Laws may be amended at any Annual Session by a majority vote of all the delegates present at the Session, after the amendment has lain on the table for one day.

Upon the adoption of these By-Laws all previous by-laws are thereby repealed.

In Memoriam

Floyd W. Burns

1876-1937

THE death of Dr. Floyd W. Burns occurred on January 20, 1937 at his home in Saint Paul. He had been in poor health for several months.

Born in Panora, Iowa, in 1876, Dr. Burns came to Saint Paul as a boy of eleven. He attended Central High School and later the University of Minnesota, where he was a member of the Phi Beta Pi fraternity. He obtained his M.D. degree at the University of Chicago and began practice in South Saint Paul in 1901.

Dr. Burns served as a captain in the medical corps of the army during the World War and was stationed at Fort Houston, Texas, during most of his service. He was a member of Post No. 8 of the American Legion and was a Mason and member of Osman Temple Shrine.

Dr. Burns is survived by his widow, a son Robert, four brothers—Ormond of Saint Paul, William of Duluth, Dr. M. A. Burns of Milan and Ernest Burns of San Diego, California.

Dr. Burns held the esteem of his fellow practitioners in Saint Paul and was well liked by his many friends.

Oscar E. Locken

1891-1937

THE death of Dr. Oscar E. Locken of Crookston came as a shock to his many friends in the medical profession throughout the state. His death occurred at the age of forty-five on January 18, 1937, after a week's illness from pneumonia.

Born in Crookston, March 8, 1891, the son of John H. and Julia Vennevold Locken, he graduated from the Crookston High School in 1909 and four years later received his B.A. degree at St. Olaf College, Northfield, Minnesota. While at college he was president of the Student Council. Two years later he entered the University of Minnesota medical school and after graduating served an internship at the University Hospital and a year at the Mayo Clinic.

Returning to Crookston Dr. Locken began practice and was one of the founders of the Northwestern Clinic in that city. In recent years he had specialized in internal medicine.

Dr. Locken had held many executive offices. At one time he was mayor of Crookston. For six years he was health officer of that city. He had also been president of the State Sanitary Conference, the Red River Valley Medical Society and the Minnesota League of Municipalities. He was also president of the commission of the Polk and Norman County Sanatorium. Recently he had been appointed to the Social Security and Public Welfare survey of the State Planning Board.

He had served for many years as first vice president and member of the Executive Committee of the Minnesota Public Health Association.

Last year when the House of Delegates of the Minnesota State Medical Association was looking for the best qualified member to preside over their deliberations, Dr. Locken was chosen. He will be greatly missed by that body.

Surviving are his widow, the former Agnes Oppegaard of Madison, Minnesota, whom he married in 1929; a son, John; and two daughters, Helene and Mary Ellen.

Erwin W. Exley
1897-1936

DR. ERWIN W. Exley of Minneapolis died May 22, 1936, from complications following appendectomy. Taken ill when about to return home from attending a medical meeting in Boston, Dr. Exley was taken from the train at New York to the Postgraduate Hospital where the operation was performed.

Dr. Exley was born in Menasha, Wisconsin, in 1897 and received his preliminary education in the public schools there. He received the degree of B.S. from the University of Wisconsin in 1921 and his M.D. from the University of Minnesota in 1925. He served his interneship at the Miller Hospital, Saint Paul, following which he was associated with Dr. Gilbert Thomas in Minneapolis for three years. He then opened an office in Minneapolis for the practice of urology.

Dr. Exley was a member of the Hennepin County Medical Society, the Minnesota State Medical Association and the American Medical Association. He was also a member of the American Urological Society and the Alpha Kappa Kappa medical fraternity.

Dr. Exley is survived by his widow, a son and a daughter, both parents who live at Menasha, and a brother, also a physician.

Henry S. Plummer
1874-1936

DR. HENRY S. PLUMMER, scientist, physician, a member of the staff of The Mayo Clinic for thirty-five years, died at his home in Rochester, Minnesota, of cerebral thrombosis on December 31, 1936, at the age of sixty-two. Dr. Plummer was born March 3, 1874, in Hamilton, Minnesota, on the old stagecoach trail to Dubuque. He took premedical courses at the University of Minnesota, and attended Northwestern University, receiving the degree of M.D. in 1898. He then practiced in Racine, Minnesota, from 1898 to 1901. Entering the Mayo Clinic in 1901, he was a pioneer in the development of roentgenology, bronchoscopy, esophagoscopy and electrocardiography.

In the next twenty years he devoted most of his time to improving methods of general medical diagnosis, and through his efforts many discoveries of science and new laboratory procedures were incorporated into clinical research at the clinic. During this period he made a special study of disturbances of the thyroid gland, and as a result cleared up many of the previously confusing ideas on the subject of thyroid disease. His studies on the effect of iodine in the treatment of exophthalmic goiter, for example, led to an immediate reduction in the mortality in this disease from about 3 or 4 per cent, what it had been in this country, to less than 1 per cent, and the necessity for multiple stage operations was almost eliminated.

On October 4, 1904, Dr. Plummer married Daisy Berkman, daughter of Dr. and Mrs. David M. Berkman. He is survived by his wife, by his daughter, Gertrude (Mrs. James A. Thomas), and by his son, Robert.

Dr. Plummer was a member of the Minnesota State Medical Association, Olmsted-Houston-Fillmore-Dodge County Medical Society, Minnesota Pathological Society, Southern Minnesota Medical Association, Central Interurban Clinical Club, Minnesota Society of Internal Medicine, Central Society for Clinical Research, Minnesota Horticultural Society, University Club and the St. Paul Athletic Club; he was a Fellow of the American College of Physicians a Fellow of the American Medical Association, and held membership in the Association of American Physicians, Association for the Study of Internal Secretions, American Association for the Advancement of Science, American Association for the Study of Goiter, American Gastro-Enterological Association, Medical Library Association, American Association for Thoracic Surgery, American Public Health Association, Royal Society of Arts, Alumni Association of The Mayo Foundation, Sigma Xi, and Alpha Omega Alpha.

In 1933, he was elected president of the Association for the Study of Goiter, and in 1935, Northwestern University conferred on him the degree of D.Sc. (honoris causa).

A man of great mechanical genius, he had much to do with the design and construction of the new clinic building, which was erected in 1929. Many features of this building attest his ingenuity. For example, he designed the conveyors which carry records and other essential data to whatever floor they are needed, and he was responsible for the system of signal lights which facilitates the handling of patients. The clinic's auditorium on the fourteenth floor, where Staff meetings are held, was fittingly named "Plummer Hall" to honor him, not only in recognition of his contributions to medicine, but to pay tribute to his mechanical and artistic genius as well.

There seemed to be no field to which his interest did not extend. He had a great deal to do with the design and construction of the Franklin Heating Station, and so great was his knowledge of all phases of engineering that he was called in as consultant by the company that supplies Rochester and vicinity with natural gas. One of his hobbies was horticulture, and at the time of his death he was experimenting with the effects of electricity on growing plants.

A man of catholic tastes, and one widely read in fields other than medicine, he believed that physicians should not neglect the more leisurely aspects of culture, and he was instrumental in having set aside in the medical library a room where one so minded could browse in the classics.

OF GENERAL INTEREST

Dr. Norman M. Smith of Minneapolis was recently elected president of the Uptown Commercial Club.

* * *

Dr. John Amberg Haugen and Miss Phœbe Saunders, both of Minneapolis, were married February 3, 1937, in Plymouth Congregational Church, Minneapolis.

* * *

Dr. Charles E. Lyght, director of health service at Carleton College, Northfield, Minnesota, was recently elected an associate of the American College of Physicians.

* * *

Dr. S. H. Anderson, eye, ear, nose and throat specialist of Red Wing, became associated the first of the year with the firm of Drs. Johnson and Steaffens, with offices in the Red Wing Clinic.

* * *

Dr. C. L. Sherman of Luverne, president of the Southwestern Minnesota Sanatorium, was named president of the Sioux Valley Medical Association, at the annual meeting held in Sioux City, Iowa, January 20.

* * *

Dr. W. G. Benjamin, of Pipestone, was recently re-elected president of the Pipestone Civic and Commerce Association at the annual meeting of the Board of Directors.

* * *

Dr. Edwin L. Gardner, Minneapolis, who was elected a member of the American Gastroenterological Association last year, has been recently notified of his election to the International Society of Gastroenterology.

* * *

The village of Lismore, Minnesota, is looking forward to having a resident physician soon. Dr. A. W. Pasek of Duluth, a graduate of the University of Minnesota, has announced that he will open an office there.

* * *

Miss Kathleen Keefe and Dr. Florian Baumgartner, both of Minneapolis, were married on January 11 in Incarnation Church, and will make their home in Minneapolis. Dr. Baumgartner will be graduated in June from the University of Minnesota School of Medicine.

* * *

Dr. Charles N. Spratt of Minneapolis addressed the King County Medical Society in Seattle, Washington, on January 18. On Tuesday, January 19, Dr. Spratt presented his films on Eye Surgery before the Puget Sound Academy of Ophthalmology.

* * *

Dr. E. L. Gardner and Dr. R. S. Ylvisaker announce their association for the practice of internal medicine with offices at 1629 Medical Arts Building, Minneapolis. They will give special attention to the diagnosis and treatment of gastro-intestinal disease.

At the annual meeting in Saint Paul, January 12, the Minnesota State Board of Health re-elected its present officers. They are Dr. Frederick Bass, University of Minnesota engineering professor, president; A. S. Milinowski, Saint Paul, vice president, and Dr. A. J. Chesley, secretary and executive officer.

* * *

A series of six weekly lectures on health was opened at the Minneapolis Y.M.C.A. Monday evening, January 4, with a talk on "Diet" by Dr. W. A. O'Brien of the University of Minnesota. Other speakers in the series are Dr. M. J. Shapiro, Dr. Donald A. Dukelow, Dr. A. G. Wethall, Dr. J. A. Myers and Dr. S. Alan Challman.

* * *

The Extension Division of the University of Minnesota announces a lecture and demonstration course in x-ray diagnosis to be given by Dr. Leo G. Rigler and his associates at the University Hospital beginning Thursday, February 11, from 6:20 to 8:00 p.m. and continuing once each week for sixteen weeks. Anyone interested should communicate with the Extension Division, University of Minnesota.

* * *

Dr. N. O. Pearce was recently named president of the Hennepin County Tuberculosis Association. For the past four years he has been chairman of the medical committee of the association and, as a member of its board of directors, he has been actively interested in the tuberculosis prevention program.

Dr. Stephen Baxter was elected first vice president; Dr. W. A. Aurand, second vice president; Mrs. Sylvester Koontz, secretary (re-elected) and W. P. Christian, treasurer.

* * *

Honored by the Rockefeller Foundation, Dr. Kenneth F. Maxcy, professor and head of the department of preventive medicine and public health at the University of Minnesota, has been chosen one of the scientific directors of the International Health Division of the Foundation. The appointment, for a three-year term, became effective January 1. Dr. Maxcy joined the University staff last fall, coming from the University of Virginia to succeed Dr. Harold S. Diehl, who was promoted to the post of dean of medical sciences.

* * *

Information has been received from a member at Brooten, Minnesota, that last September a man who signed his name "E. M. Giess" and claimed to represent the Good News Agency of Brookings, South Dakota, spent about ten days at Brooten selling magazine subscriptions. When no magazines were received a letter was sent to the above agency but was later returned unclaimed. Apparently no such agency exists and warning is therefore given. Agent Giess is a cripple thirty-five to forty years of age, about 4 feet

10 inches in height and weighs about 100 pounds. His teeth are poor. There is considerable wasting of the muscles of his arms and legs and his speech is almost inarticulate.

* * *

Announcement has been made that Dr. Charles B. Lenont, dean of Virginia's medical fraternity, and Dr. Edward N. Peterson of Eveleth, will unite in the establishment of a medical clinic in Virginia, to be known as the Lenont-Peterson Clinic. The clinic will open February 1, in remodeled and newly equipped quarters in the Lenont Hospital Building. When fully completed, sometime in May, an additional investment of between $25,000 and $30,000 will be represented. The complete medical staff has not yet been selected, and it is the plan to build up the personnel gradually, with trained men in all departments of medicine and surgery. The clinic, in addition to the recently opened $300,000 Municipal Hospital, will establish Virginia as an important medical center for Northern Minnesota. Dr. Lenont is chief-of-staff of the new hospital.

* * *

A program of medical lectures arranged by the Minnesota branch of the American Association of Medical Social Workers for the winter season opened Friday evening, January 22, with a talk on "Medical Ethics for Social Workers" by Mr. James Baker, executive secretary of the Hennepin County Medical Society. On February 12, Miss Christ of the University Hospital Social Service Department, will present a paper on "Syphilis in the Eye." Dr. Erling Hansen will discuss the paper. Dr. Hilleboe, director of tuberculosis division, State Board of Control, will speak February 26 on the subject "Crippled Children Under the Social Security Act." "Orthopedic Problems" will be presented by Dr. E. T. Evans on March 12, and Dr. E. M. Rusten will discuss the question of "Allergy" on March 26. Dr. Bernard Watson of the University Student Health Service will present the subject "Hyperthyroidism and Diabetes" at the meeting of April 9.

All lectures will be given at 4:30 p.m. Fridays in the lecture room of the Hennepin County Medical Society headquarters, 2000 Medical Arts Building, Minneapolis.

Questions Requested

A symposium on obstetrics and gynecology has been arranged for the 84th Annual Meeting of the Minnesota State Medical Association at the St. Paul Auditorium, May 3, 4, and 5, by the Committee on Scientific Assembly.

Practical problems of the practitioner are to be principally considered by the symposium speakers at this session. Members are accordingly requested by the committee to send to state headquarters immediately any questions they would like to have discussed and answered. The questions will be incorporated in the talks given by the symposium speakers.

HOSPITAL NEWS

Dr. L. H. Cady has been named chairman of the medical staff of St. Andrew's Hospital, Minneapolis, to succeed Dr. Walter Ude, retired.

* * *

Dr. Carl G. Swendseen has been named chief of staff at Swedish Hospital, Minneapolis. Other officers will be Dr. Oliver H. Peterson, vice chief; Dr. Earl H. Dunlap, secretary, and Dr. Charles R. Drake, treasurer.

* * *

At the forty-fourth annual meeting of the Executive Board of Asbury Hospital, held on January 17, speakers included Bishop J. Ralph Magee of St. Paul, Dr. R. C. Webb, retiring chief of staff of the hospital, and Dr. H. B. Dornblaser, incoming chief of staff.

* * *

St. Mary's Hospital, Minneapolis, and St. Joseph's Hospital, Saint Paul, recently joined the Minnesota Hospital Service Association. Plans are also being made for the establishment of a regional branch of the Association in Wadena, it has been announced.

* * *

Victor S. Lindberg, assistant to Rev. L. B. Benson, superintendent of Bethesda Hospital, Saint Paul, for the past three and a half years, has been appointed superintendent of the Swedish-American Hospital at Rockford, Illinois, where he began his duties February 1.

* * *

At the annual meeting of the Minneapolis Hospital Council recently held, Dr. F. O. Hanson, Superintendent of Swedish Hospital, was re-elected president. Others returned to office were Harry Brown, Northwestern Hospital, vice president; Sister Anna Berglund, Deaconess, treasurer; and Rebecca Peterson, St. Andrews, secretary.

* * *

The fiftieth anniversary of the founding of Maternity Hospital, Inc., was observed at the annual meeting of the corporation on January 20. Mrs. John C. Benson was named president; Mrs. L. A. Page, first vice president; Mrs. Lester R. Badger, secretary; Mrs. George B. Clifford, treasurer. The program included a reading of the report of the first annual meeting of the hospital, and a tribute to Mrs. Martha G. Ripley, founder of the hospital.

* * *

New Radiological Department
The Charles T. Miller Hospital

The Charles T. Miller Hospital of Saint Paul has just completed plans, and construction will begin in the very near future, of an addition and alterations to the first and second floors of the northwestern wing of the hospital, for the installation of a supervoltage, 1,200,000 volt, constant potential x-ray therapy machine which is being built by the Kelley-Koett X-ray Company of Covington, Kentucky, for Dr. Edward Schons and Dr. J. P. Medelman, who will be the directors of

the radiological department. The building and machines will be completed and ready for operation June 1, 1937.

This supervoltage equipment will be the largest and most advanced of its kind, and will have the capacity of treating four patients at a time. It will be used primarily for the more adequate and efficient treatment of deep-seated tumors. The very high voltage and filtration used will yield a radiation of practically gamma ray quality and of unlimited quantity in comparison with external irradiation with large radium packs, available, for economic reasons, in only a few institutions for treatment of a limited number of patients. Lower voltage equipment will also be installed and will be used as heretofore, where that type of irradiation is indicated, as will also radium.

Today, some hospitals in this country have supervoltage machines in operation, ranging in capacity from 400,000 to 800,000 volts. Reports from these medical centers, such as Harper Hospital, Detroit, Memorial Hospital, New York, Lincoln General Hospital, Lincoln, Nebraska, Northwestern Hospital, Minneapolis, University of Chicago Clinics and Mercy Hospital, Chicago, and others, are exceedingly favorable of the results obtained. Still better results are, therefore, hoped for from the radiation which will be obtained from the new equipment.

The x-ray tube, constructed of indestructable porcelain and metal, in which electrons will bombard a lead-coated copper electrode to create the radiation necessary for treatment, is 27 feet long. It is protected by four inches of lead to prevent the escape of radiation except where it is desired.

The target of the tube, or the x-ray producing electrode, will be constructed of copper, five inches in diameter and one millimeter thick, covered by a thin layer of lead. Lead is used because of its high atomic weight. The electron streams bombarding it will produce a more penetrating radiation than with metal of less atomic weight. The copper and lead target is backed up with a cooling jacket. Oil is used to cool the target; water to cool the oil.

The operator will be protected from radiation by very heavy concrete walls lined with lead. Very adequate measuring equipment will provide for constant control of the quality and quantity of radiation being used. A two-way microphone loud speaker communication system will be provided to permit conversation with the patient, and a periscope will permit observation of the patient during treatment.

The new addition to the Miller Hospital, which is especially designed to house the supervoltage equipment, required skilled engineering. It was, therefore necessary for the architects, Clarence H. Johnston, Inc., of Saint Paul, to work in close coöperation with the engineers of the Kelley-Koett X-ray Company, in designing and drawing the plans. The Kelley-Koett X-ray Company is a pioneer in the development of x-ray equipment, having been prominent in the field since the early days of the x-ray. The plans were reviewed with Dr. Lauriston S. Taylor of the U. S. Bureau of Standards some time ago, before final adoption.

REPORTS AND ANNOUNCEMENTS OF SOCIETIES

Medical Broadcast For February

The Minnesota State Medical Association Morning Health Service

The Minnesota State Medical Association broadcasts weekly at 2:30 o'clock every Thursday afternoon over Station WCCO, Minneapolis and Saint Paul (810 kilocycles or 370.2 meters).

Speaker—William A. O'Brien, M. D., Associate Professor of Pathology and Preventive Medicine, Medical School, University of Minnesota.

The program for the month will be as follows:
February 4—As We Grow Older.
February 11—First Aid.
February 18—Colitis.
February 25—Loss of Teeth.

State Meeting

A scientific exhibit section of exceptional interest to medical men of the Northwest has been assembled for the 84th Annual Meeting of the Minnesota State Medical Association to be held at the St. Paul Auditorium May 3, 4 and 5.

The Northwest Industrial Medical Conference which occupies an entire day of the meeting program has prompted a unique exhibit group on industrial medicine in which the United States Public Health Service, the Division of Labor Standards, the United States Navy, Safety Engineer; the United States Army, Surgeon General and the Aviation Department, and the State Department of Health of Connecticut probably will participate.

The stellar feature of the exhibits will be provided by Dr. A. E. Jenks of the University of Minnesota Sociology Department and Dr. C. F. Jackson, head of the Anatomy Department of the University Medical School. It will consist of Minnesota's pre-historic girl, inhabitant of the Pleistocene Age discovered by Dr. Jenks, and an exhibit on comparative anatomy provided by Dr. Jackson.

Other important exhibitors include: Dr. F. H. Krusen of Rochester and Dr. M. E. Knapp of Minneapolis who will combine to present an extensive exhibit and demonstration of physical therapy; the Minnesota-Dakota Orthopedic Club that will exhibit and demonstrate fracture treatment; the Minnesota Academy of Ophthalmology and Otolaryngology; Dr. L. F. Hawkinson of Brainerd who will have a new exhibit on endocrinology; the American Medical Association which will exhibit a variety of subjects including cosmetics, obesity, medical economics; the Cancer Committee which will combine with the American Society for the Control of Cancer to have actual laboratory examination of specimens at the meeting; the Children's Hospital and the Gillette State Hospital; Dr. Hamline Matson, Minneapolis, who will exhibit

on hand infections; the Mayo Clinic, the University of Minnesota and many others.

An hour of each morning and afternoon session will be devoted exclusively to exhibits and scientific demonstrations.

Technical exhibitors who have already purchased exhibit space for the meeting follow. Members are especially urged to patronize them in recognition of their coöperation in the meeting.

Abbott Laboratories
North Chicago, Ill.

American Hospital Supply Corp.
Chicago, Ill.

C. F. Anderson Co., Inc.
Minneapolis, Minn.

Ayerst, McKenna & Harrison
Montreal, Canada

Bilhuber Knoll Corp.
Jersey City, N. J.

Boehm Surgical Instrument Corp.
Rochester, N. Y.

The Borden Company
New York, N. Y.

Brown & Day, Inc.
St. Paul, Minn.

Burroughs Wellcome & Co.
New York, N. Y.

Bush X-Ray Company
Minneapolis, Minn.

Coca Cola Company
Atlanta, Ga.

Davies Rose & Co.
Boston, Mass.

DeVilbiss Company
Toledo, Ohio

Dictograph Products Co.
New York, N. Y.

Encyclopedia Britannica, Inc.
Minneapolis, Minn.

H. G. Fischer & Co.
Chicago, Ill.

Ford Motors, Twin City Branch
St. Paul, Minn.

General Electric X-Ray-Corp.
Minneapolis, Minn.

General Foods Sales Co., Inc.
New York, N. Y.

General Heat & Air Engineers
Minneapolis, Minn.

Gerber Products Division
Fremont, Michigan

H. J. Heinz Co.
Pittsburgh, Pa.

Horlick's Malted Milk Corp.
Racine, Wisc.

Kellogg Co.
Battle Creek, Mich.

I. Kessell & Co.
St. Paul, Minn.

Lea & Febiger, Publishers
Philadelphia, Pa.

Lederle Laboratories
New York, N. Y.

Lepel High Frequency Laboratories
New York, N. Y.

Libby, McNeill & Libby
Chicago, Ill.

J. B. Lippincott Co.
Philadelphia, Pa.

Mead-Johnson Co.
Evansville, Indiana

Medical Protective Co.
Wheaton, Ill.

Mellin's Food Co.
Boston, Mass.

Merck & Co.
Rahway, N. J.

Wm. S. Merrell Co.
Cincinnati, Ohio

Middlewest Instrument Co.
Chicago, Ill.

C. V. Mosby Book Co.
St. Louis, Mo.

V. Mueller & Co.
Chicago, Ill.

Pengelly X-Ray Co.
Minneapolis, Minn.

Petrolagar Laboratories
Chicago, Ill.

Philip Morris & Co., Ltd.
New York, N. Y.

Physicians & Hospitals Supply Co.
Minneapolis, Minn.

W. B. Saunders Co.
Philadelphia, Pa.

Schering Corp.
Bloomfield, N. J.

Upsher Smith Co.
Minneapolis, Minn.

Sonotone-Minnesota Co.
Minneapolis, Minn.

E. R. Squibb & Sons
New York, N. Y.

Standard X-Ray Sales Co.
St. Paul, Minn.

U.S. Hospital Supply Co.
Minneapolis, Minn.

Universal Products Corp.
Pottstown, Pa.

Winthrop Chemical Co.
New York, N. Y.

Clay-Becker County Society

The following are the officers of Clay-Becker County Medical Society for 1937:

President—A. R. Ellingson, Detroit Lakes.
Vice President—E. K. Ingebrigtson, Moorhead.
Secretary-treasurer—L. H. Flancher, Lake Park.
Delegate—C. W. Simison, Hawley.
Alternate—L. H. Rutledge, Detroit Lakes.

Rice County Society

At the meeting of the Rice County Medical Society held in the Faribault Clinic Rooms, on Thursday, January 28, motion pictures provided through the courtesy of Davis & Geck, Brooklyn, N. Y., were shown on the following subjects:

1. Subtotal Thyroidectomy
2. Perineal Prostatectomy for Benign Hypertrophy
3. Montgomery-Simpson Suspension of Uterus
4. The Latzko Extraperitoneal Cesarean Section

Scott-Carver County Society Meets with Minnesota Valley Dental Study Club

The Scott-Carver County Medical Society, in conjunction with the Minnesota Valley Dental Study Club, held a meeting at Mudbaden Sanitarium, Monday, January 11, 1937. The meeting was devoted to a discussion of economic and legislative matters of interest to both groups.

Speakers for the Scott-Carver Society were Dr. L. L. Sogge of Windom and Mr. Manley Brist of Saint Paul. Guest speakers from the Dental Club were Dr. Clayton Swanson and Dr. Louis Weiss, both of Minneapolis.

Steele County Society

Dr. J. F. Schaefer was elected president of the Steele County Medical Society for the coming year. Dr. Benedick Melby of Blooming Prairie was chosen vice president and Dr. C. T. McEnaney of Owatonna, secretary-treasurer.

Waseca County Society

Dr. William Bernstein of New Richland was chosen president of the Waseca County Medical Society for 1937. Dr. R. C. Hottinger of Janesville was named vice president and Dr. George H. Olds of Waseca, secretary-treasurer.

Washington County Society

The following are the officers of Washington County Medical Society for 1937:

President—J. W. Stuhr, Stillwater.
First Vice President—R. P. Ewald, Newport.
Second Vice President—E. V. Strand, Bayport.
Secretary-treasurer—E. S. Boleyn, Stillwater.
Delegate—E. S. Boleyn, Stillwater.
Alternate—W. R. Humphrey, Stillwater.
Censor for three years—D. Kalinoff, Stillwater.

Winona County Society

At the annual meeting of the Winona County Medical Society held Monday evening, January 4, in Winona, the following officers were elected for 1937:

President—Dr. A. E. Meinert, Winona.
Vice President—Dr. Robert Tweedy, Winona.
Secretary-treasurer—Dr. I. W. Steiner, Winona.

WOMAN'S AUXILIARY

Mrs. E. M. Hammes, *President*,
1456 Summit Avenue, Saint Paul
Mrs. A. A. Passer, *Editor, Press and Publicity*, Olivia

St. Louis County Auxiliary

Gold and silver appointments decorated the tables for the dinner dance given by the members of the St. Louis County Medical Society and its Auxiliary in the ballroom of the Spalding Hotel in Duluth in December. The occasion was the fiftieth anniversary of the founding of the St. Louis County Medical Society and the twenty-fifth anniversary of the organization of the Auxiliary.

Arrangements were completed by Mrs. Robert S. Forbes, president of the Auxiliary, and Dr. Harry Klein, president of the County Society.

The program included an address by Prof. Herbert Heaton of the University of Minnesota on "1886 and All That." Dr. O. W. Parker of Ely gave a brief history of the range activities and Dr. Klein gave a résumé of the history of the county group. Dr. Malcolm Gillespie presented a pictorial history of the County Society.

Mrs. Robert S. Forbes, president of the Auxiliary, announced the addition of a ten dollar cash prize to the awards being made by the state group to winners in the statewide high school public speaking contest sponsored by the Minnesota Public Health Association and its affiliated associations. The cash award is made by the St. Louis County Auxiliary.

Hennepin County Auxiliary

A first prize of ten dollars and a second prize of five dollars were given to the winners in Senior High Schools and a similar prize was awarded to the winners in Junior High Schools by the Hennepin County Auxiliary in addition to the state prizes in the public speaking contest.

The regular meeting of the Hennepin County Auxiliary was held in the Medical Arts Library on December 4 in the form of a Christmas party.

Mrs. J. M. Neil and Mrs. A. N. Bessesen, Jr., arranged the joint party of the Hennepin County Medical Society and its Auxiliary held in the Medical Arts Library on January 18. A smörgåsbord was held at 6:30 p.m. followed by a program of cards. Members of the Auxiliary wore Swedish costumes.

The sale of articles made by patients at Glen Lake Sanitarium and conducted by the Hennepin County Auxiliary was an outstanding success. The receipts of the three-day sale were $1,066.20. This is the fourth consecutive year of this sale and each year receipts have increased.

Renville County Auxiliary

The annual Christmas party of the Renville County Auxiliary was held at the High School in Olivia, Tuesday evening, December 1. After the program the members were entertained at lunch by the Medical Society.

Ramsey County Auxiliary

Mrs. Mark Ryan, chairman of the year book committee for the Ramsey County Auxiliary, distributed the books at a meeting of the Auxiliary held at the home of Mrs. Joel Hultkrans, Saint Paul. Three papers by school children were read on the subject "The Thirty Years' Fight Against Tuberculosis." Miss Bonnie Jean Kelly was presented with a silver trophy by the Auxiliary for the best essay. The contest was open to all Ramsey county high school students. Judges for Ramsey County were Mrs. Warner Ogden for the Auxiliary; Dean Charles S. Templer of Hamline University; and Dr. Thomas E. Broadie, superintendent of Ancker Hospital.

Mrs. Herman Kesting is chairman of the committee for the series of play reviews given by Mrs. Arthur A. Stewart for members of the Ramsey County Auxiliary. Six popular plays will be reviewed by Mrs. Stewart on Tuesday mornings in the medical library rooms of the Lowry Medical Arts Building.

Scott-Carver County Auxiliary

The regular meeting of the Scott-Carver Auxiliary was held at the home of Mrs. H. M. Juergens of Belle Plaine. Mrs. W. F. Maertz of New Prague is president of the Auxiliary and conducted the business meeting.

* * *

Mrs. E. M. Hammes, president of the State Medical Auxiliary; Mrs. James Blake, finance chairman of the National Auxiliary, and Mrs. J. F. Norman, president-elect of the State Auxiliary, attended the National Board Meeting held in Chicago in November.

Tetrachlorethylene.—"Tetrachlorethylene contains not less than 99 per cent and not more than 99.5 per cent of CCl_2:CCl_2, the remainder consisting of alcohol." N. F. Tetrachlorethylene has been shown to be a useful anthelmintic for the treatment of hookworm infestation. It is the consensus of the investigators that tetrachlorethylene is less toxic than carbon tetrachloride and at least as efficacious as the latter drug. Untoward reactions are rare, but giddiness, vomiting and drowsiness have been reported in some cases.

* * *

Pernoston.—Butyl -β-bromallyl barbituric acid.—Pernoston differs from barbital (diethylbarbituric acid) in that both of the ethyl groups of the latter are replaced, one by a (normal) secondary butyl group, and the other by a substituted brominated allyl group. The actions and uses of Pernoston are essentially similar to those of barbital, but Pernoston is more active than barbital and is used in correspondingly smaller doses. It is used in combating insomnia due to emotional strain and nervous instability. In therapeutic doses it is said to produce no demonstrable toxic effects on the heart, lungs, blood vessels and kidneys. It is supplied in the form of tablets, 3 grains.—*New and Non-Official Remedies.*

PROCEEDINGS of the MINNESOTA ACADEMY of MEDICINE

Meeting of December 9, 1936

The regular monthly meeting of the Minnesota Academy of Medicine was held at the Town and Country Club on Wednesday evening, December 9, 1936. The meeting was called to order by the President, Dr. Thomas S. Roberts, at 8 p. m.

There were forty-seven members and one guest present.

Minutes of the November meeting were read and approved.

The Secretary read a letter of resignation from Dr. John T. Rogers, a past President of the Academy. The secretary stated that the Executive Committee had voted and recommended to the Academy that Dr. Rogers' name be placed on the Honorary Membership list. This recommendation was passed unanimously.

The following officers were elected for 1937:

President .....Dr. E. M. Jones, St. Paul
Vice-President ..Dr. R. T. LaVake, Minneapolis
Secy.-Treas. .... Dr. Albert Schulze, St. Paul

Dr. Roberts asked the newly-elected President to take the Chair, and Dr. Jones expressed his appreciation of the honor accorded him in this election.

The scientific program followed.

EPISCLERITIS AND ITS RELATION TO DISEASE OF FEMALE PELVIC ORGANS

WILLIAM L. BENEDICT, M.D.
Section on Ophthalmology, The Mayo Clinic
Rochester, Minnesota

Dr. Benedict read his Inaugural Thesis on the above subject. (To be published in MINNESOTA MEDICINE.)

Abstract

Episcleritis and scleritis appear in various forms as acute, intermittent or chronic affections of one or both eyes. The disease attacks only adult persons and is more common in women than in men. The superficial forms and some of the intermittent forms of the disease are not harmful to sight even though they persist over many years. The deeper forms of the disease affecting the sclera and uvea lead to permanent changes in the coats of the eyeball. Some forms are very painful during the stage of inflammation. Repeated attacks of scleritis lead to thinning of the sclera, the appearance of slate-colored areas in the anterior sclera where inflammatory nodules have been situated, staphylomata in the ciliary zone, and sclerosing keratitis. Through changes in the uvea, the lens and vitreous become cloudy and in some cases secondary glaucoma leads to blindness.

The etiology of the disease has been attributed to tuberculosis, syphilis, gout, leprosy, focal infection, and disturbances of menstruation. It has long been known that episcleritis is associated with uterine disorders and is prone to occur in adult females who are subject to disturbed menstruation. Histopathologic studies have confirmed the diagnosis of tuberculosis in many eyes enucleated because of grave effects of severe scleritis. Some oculists have stated that nearly all cases of episcleritis and all cases of nodular scleritis are due to tuberculosis, but neither pathologic examination nor clinical experience offers adequate confirmation of this assumption.

Studies of a series of cases of scleritis in women in whom a relation between the attacks and disturbances of menstruation could be established showed that the cervix and uterus were foci of infection. Bacteriologic studies revealed a green-producing streptococcus as the offending organism in all cases. In cases where this relationship could be established, attempts to correct the uterine disorder were made. In some cases the cervix was cauterized; in others, hysterectomy was done. Improvement in the eye condition invariably followed operation. Recurrences were rare and in most instances mild.

Discussion

DR. FRANK BURCH, St. Paul: I am sure I speak not only for the ophthalmological group of this Academy but for all the members in welcoming Dr. Benedict into the organization, and also thanking him for again emphasizing the relation of eye diseases to general diseases. Dr. Benedict has made a real contribution along several different lines establishing such relationships, particularly the relation of prostatitis to iritis. In this thesis he has added the fact that episcleritis is not only more prevalent in women, but that it has a definite cause in pelvic infections. Most of us do not see cases of episcleritis frequently. In other intraocular inflammations, as well as in episcleritis, we are beginning to learn in our studies of their etiology that we sometimes have to go far afield in order to direct the proper treatment. Practically all of our treatment heretofore was local, aided by non-specific vaccines, foreign protein therapy, etc. From my own experience, where this relationship of episcleritis with pelvic infection has been established, I had not been able to get any results from vaccines. Patients were improved or cured when referred to the gynecologist and received proper treatment. I believe Dr. Benedict's thesis is an important contribution and that he has established a rational basis for treatment of episcleritis.

DR. C. N. SPRATT, Minneapolis: Dr. Benedict has not mentioned the names of two men who have done considerable work on the etiology of scleritis. Both of these, Verhoeff and Stock, came to the conclusion that it was a form of tuberculosis. While I was house officer under the former at the Massachusetts Eye and Ear Infirmary, and at Freiberg where I have seen the work of the latter, I was much impressed with their findings. Verhoeff had done considerable microscopic work and in addition to this, Stock had injected the

ear vein of rabbits with cultures of tubercle bacilli and had found that lesions of the choroid, uvea and sclera had followed which were very similar to conditions observed in humans. In 1911, I reported a series of cases of scleritis treated with tuberculin, before the Minnesota State Medical Association. All of these had been given, previous to treatment, a focal, general and local reaction to tuberculin. All of these patients recovered. Sometime after this a patient under tuberculin treatment had a lighting-up of a pulmonary condition, and since then I have discontinued its use in all cases. Verhoeff likewise has discontinued the use of tuberculin and relies entirely upon hygienic measures.

I rely entirely upon the application of the Shahan thermophore in the treatment of these cases. One application of this instrument at a temperature of 145° F. for one minute has been followed by cure within ten days to two weeks. This temperature causes no permanent damage to the tissues of the eye. Recurrences do occur in a few cases and it would not seem that hysterectomy would be called for. Vaccines and foreign protein therapy have not been employed in any cases under observation.

DR. BENEDICT (in closing): One cannot consider the diseases of scleritis and episcleritis without recognizing several different forms of the disease. Some individuals who are neurotic in temperament have a mild episcleral injection, sometimes diagnosed as conjunctivitis, which lasts for a few days and then disappears. That condition is known as episcleritis fugax. It probably is not due to infection. It has been assumed that it is due to some endocrine disturbance. We have no pathological proof of this. There is also an episcleritis which involves only the superficial tissues of the eye and occurs in the menstrual periods. It is noted in the textbooks of Weeks, Fox, de Schweinitz and others. Exacerbations have been noted at menstrual periods or at missed menstrual periods and are interpreted as vicarious menstruation. The etiology of tuberculosis has been brought into the discussion. Some years ago Dr. Knight and I reported on two eyes which had been removed. In those two patients there was no clinical evidence of tuberculosis but the pathological picture was that of tuberculosis. We know only too well that the pathological appearance of tuberculosis is mixed so much with the pathological appearance of local granulomas and some systemic diseases that it is difficult for the pathologist to distinguish a difference.

Whether the uveal tract is involved secondarily or whether it is a coincident infection with lesions in the sclera has given rise to considerable discussion. As I said in my paper, it is not clearly established whether this is a single infection which is transmitted to the uveal tract or whether it is a separate infection. The lesions in the eye are histologically similar to tuberculosis and frequently attributed to focal infection, and our studies have shown that a green-producing streptococcus will produce such a lesion. We have been unable to find any bacterium aside from streptococcus which would produce such a lesion.

I have at hand case histories collected during the past twenty years—37 cases in all—in which amputation of the cervix or hysterectomy was performed. In no case was hysterectomy performed only because of infection in the eye itself; but where there was evidence of uterine infection. The infection in the sclera usually disappeared within three days from the time the operation was performed and it never recurred.

I have used the thermophore for its local effect to reduce inflammation in the eye. At temperatures of 140° to 160° the thermophore is kept in contact with the sclerotic nodule long enough to produce local reaction without necrosis. After a few days the inflammatory lesion will disappear, but that is by no means a cure. So long as the source of the disturbance has not been removed there is no question but what recurrences will take place though at irregular intervals.

Peculiarly enough, our clinical observation has shown that all through the child-bearing period there may be no evidence of scleritis, particularly during the periods of pregnancy and lactation.

It is impossible to conceive of episcleritis as being a local disease of the eye. Simply to treat the local disease (and there are many ways of getting rid of the local reaction) has absolutely no influence on the cause of the disease; and to assume that getting rid of the local lesion in any way gets rid of the origin of the disease is to blind one's self to the facts. Episcleritis is evidence of disease somewhere else in the body. The clinical observation that inflammation of the eye subsided after the removal of an infected uterus led us to believe that here was a source of infection that was just as potent as infection of the teeth. Cultures of teeth, tonsils and pelvic organs (uterus in women and prostate in men) always gave us the same type of streptococcus. Therefore, we had reason to believe that if a woman had recurrent attacks of episcleritis and if there was no question about the virulence of streptococci in the pelvic organs, we were justified in removing the uterus.

AGRANULOCYTOSIS

ALFRED HOFF, M.D.
Saint Paul

Dr. Alfred Hoff, of St. Paul, read a paper on the above subject. Slides and charts were shown and cases reported. (To be published in MINNESOTA MEDICINE.)

Abstract

In 1922 Werner Schultz described a highly fatal syndrome which he regarded as a new and distinct clinical entity and for which he proposed the term "Agranulocytosis." Subsequent terminology by various writers included Agranulocytic Angina, Idiopathic Neutropenia, Malignant Neutropenia, and Primary Granulocytopenia.

It occurred mostly in elderly women and was characterized by necrotizing lesions in the mouth, pharynx, rectum and vagina, and was associated with fever, marked prostration and a profound leukopenia with complete or near complete absence of granulocytes in the circulating blood, but with little if any anemia or reduction in the blood platelets.

Since then much discussion has arisen as to whether or not it really constituted a new or a distinct clinical entity.

Surveys of the medical literature by numerous writers—among whom especially to be mentioned are Thomas Fitz-Hugh, Jr., and Roberts and Kracke—indicate that prior to his original description there were only three reports which at the present time would be classified as agranulocytosis; (1) by Brown in 1902, "A Fatal Case of Acute Primary Pharyngitis with Extreme Leukopenia;" (2) one by Schwartz in 1904, "A Case of Extreme Leukopenia;" and (3) one by Tuerck in 1907, "Septic Disease with Atrophy of the Entire Granulocytic System."

According to Fitz-Hugh, Brown believed that his case was identical with those of Phlegmon of the Pharynx reported by Senator in 1888.

Kracke and Parker stated that "it was responsible for more than 1500 deaths in the United States alone in the three year period ending in 1934." They give a comprehensive review of the literature in an excellent article appearing in the Journal of the American Medical Association (Sept. 21, 1935) entitled "The Relationship of Drug Therapy to Agranulocytosis." The salient features in the etiological approach were summarized and the accumulative evidence incriminating admidopyrine as a causative factor given.

Leukopenia and granulopenia are frequent accompaniments of many diseased states, such as the leukopenic phase of an acute leukemia, pernicious anemia, aplastic anemia and certain infectious diseases such as typhoid and typhus fever, et cetera. However, in these the clinical features may be and often are distinctive and serve to make diagnosis possible.

Fitz-Hugh and Krumbhaar in 1932 reported the pathological changes found in the bone marrow in three fatal cases and stated that the marrow of the bones examined in one case contained active hemopoietic areas filled with myelocytes, promyelocytes and myeloblasts while the peripheral blood contained only 200 w.b.c. per cu. m.m. In the other two cases there was likewise absence of myeloid aplasia. They suggested a condition of maturation arrest as an explanation for the paucity of the circulating granulocytes.

Henry Jackson, Jr., in a recent article, agrees with this viewpoint and in addition to twenty-seven of his own cases coming to autopsy cites eleven cases analyzed by Custer in which "there are marked proliferation of the myeloblasts with failure of these cells to mature, while the other elements of the bone marrow were undisturbed."

Therefore, neither marked anemia nor thrombopenia are features of this disease. If one permits a severe anemia or hemorrhages in the skin to enter into the clinical picture, the diagnosis of agranulocytosis becomes hopelessly confused with other types of bone marrow insufficiently and especially with the acute phase of aleukemic leukemia whose symptoms in every other respect may be identical.

The present concept of agranulocytosis holds that it is due to a depressed condition of the bone marrow in which a selective failure of the myelocytic function occurs causing a complete or a near complete disappearance of the granulocytes in the blood stream. The granulocytes protect the body against bacterial invasion and with their disappearance active immunity is lost and local bacterial invasion takes place in the form of necrotic lesions in the mouth, pharynx and rectum. General septic invasion results unless timely granulocytic recovery takes place. However, general sepsis may be so abrupt as to preclude the possibility of timely granulocytic response, thus resulting in the acute fulminant type with an invariably fatal outcome.

Four cases were presented with one recovery and three deaths. Autopsy was obtained in one case.

Slides were presented, showing the course, with frequent w.b.c. and differential counts, as well as more infrequent r.b.c. counts and Hb. determinations and the treatment employed.

Two cases followed the regular prolonged use of allonal. One case that died was in the hospital for a different ailment and developed an acute fulminant agranulocytosis after the daily use of two allonal tablets for thirty-one days. One case followed the use of dinitrophenol.

Allonal, according to its manufacturers, is allyl isopropylbarbituric acid chemically fused with amidopyrine in the proportion of 1:1⅔. It enjoys considerable popularity as a pain relieving and sleep inducing drug, both among physicians and the laity, and in consequence is extensively used. Ordinarily it may be administered with unquestioned safety. I had one patient who took two, sometimes three, tablets every night for four years without demonstrable injury. But the accumulated evidence against amidopyrine-containing drugs is such as to warrant the statement that its prolonged use in the occasional sensitized individual may result in agranulocytosis and death. There is no exact method for accurately determining such sensitivity and, as a result, where its use is unduly prolonged it becomes necessary to check up such patients with frequent total and differential white blood cell counts for evidence of leukopenia and granulopenia and also to exert our best efforts to prevent its indiscriminate use among the laity.

Discussion

Dr. C. E. Connor, St. Paul: Our present interest in this entity dates from 1922 when Schultz described it as we have it today. Dr. Pepper, of the University of Pennsylvania, recently gave an historical résumé of which he mentioned the fact that MacKenzie in 1880 referred to Gübler as having first described agranulocytosis in 1857; Trousseau, in 1865, differentiated it from other anginas. Pepper thought they were describing what we know today as agranulocytosis; if so, the disease was lost sight of until Schultz brought it to our attention again.

The differential diagnosis from other types of malignant neutropenia, particularly acute leukemia, Vincent's angina, acute streptococcic sore throat and diphtheria, depends largely on laboratory methods, especially the differential blood counts and smears and cultures of the throat. There is nothing pathognomic about the local lesion.

DR. HOFF (in closing): This disease seems to be more of a private hospital disease than a city hospital disease. In a service of about 25 years at the Ancker Hospital I cannot recall ever having seen a case of agranulocytosis in that hospital. Possibly public hospital patients do not indulge in prolonged self-medication with the drugs of this group. Allonal is being used a great deal and this possibility of doing damage should be recognized.

The meeting adjourned.

R. T. LAVAKE, M.D.
Secretary.

Meeting of January 13, 1937

The Annual Meeting of the Minnesota Academy of Medicine was held at the Town and Country Club on Wednesday evening, January 13, 1937. The meeting was called to order at 8 o'clock by the president, Dr. E. M. Jones.

There were fifty-three members and four guests present.

Reading of the minutes and all other business was dispensed with and Dr. Jones turned the meeting over to the essayist of the evening.

DR. THOMAS S. ROBERTS, Minneapolis, retiring president, then said he would depart from the usual custom of addressing the Academy on some scientific subject and talk about his "hobby" instead. Dr. Roberts gave a most interesting and entertaining "Review of the Bird Life of Minnesota," illustrated with slides and colored motion pictures.

The meeting adjourned.

A. G. SCHULZE, M.D.
Secretary.

The Physician and the Traffic Problem

Lowell S. Selling, Detroit (*Journal A. M. A.*, Jan. 9, 1937), is of the opinion that the chief reason for criticism of the tests proposed in some states and now given to drivers in others, which should be of interest to physicians, is the fact that these tests in themselves do not separate the good from the bad driver. Many of the driving difficulties are due to emotional handicaps or arise from some temporary physical condition that is correctable or that might not occur again in the same individual during the rest of his life. Under these conditions the mere physical examination, a mere check-up of the eyesight, or a brief psychologic test, such as the Binet or some simpler test, would fail to reveal why the man under consideration had his accident or why he is a chronic law violator. Until physicians themselves give these examinations, compile data and show just where the line must be drawn between adequate and inadequate physical capacities, licensing by means of physical and mental tests will be more or less of a farce. A mere physical handicap is no contraindication to driving, and it requires the decision of an experienced and highly trained individual to make a determination. The features which the physician must consider when mapping out plans for making examinations for driver's license, or examining offenders or persons involved in accidents, from the physical and mental standpoint, are the general physical condition, the eye examination and mental deviations.

BOOK REVIEWS

Books listed here become the property of the Ramsey and Hennepin County Medical libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

PHYSICAL THERAPEUTIC METHODS IN OTOLARYNGOLOGY. Abraham R. Hollender, M.D., F.A.C.S. Associate in Laryngology, Rhinology and Otology, University of Ill. College of Medicine, etc. 442 pages. Illus. Price, cloth, $5.00. St. Louis: C. V. Mosby Co., 1937.

CARCINOMA OF THE FEMALE GENITAL ORGANS. M. C. Malinowsky and E. Quater. Translated from the Russian by A. S. Schwartzmann, A.B., M.D. 255 pages. Illus. Price, cloth, $5.00. Boston: Bruce Humphries, Inc., 1937.

THE MANAGEMENT OF OBSTETRIC DIFFICULTIES. Paul Titus, M.D. Obstetrician and Gynecologist to St. Margaret Memorial Hospital, Pittsburgh, etc. 879 pages. Illus. Price, cloth, $8.50. St. Louis: C. V. Mosby Co., 1937.

ANNUAL REPORT OF THE SURGEON GENERAL OF THE PUBLIC HEALTH SERVICE OF THE UNITED STATES. For the Fiscal Year of 1936. 158 pages. Illus. Price, cloth, $1.00. Washington, D.C.: United States Government Printing Office, 1937.

PROCEEDINGS OF THE THIRTIETH ANNUAL CONVENTION OF THE ASSOCIATION OF LIFE INSURANCE PRESIDENTS. 251 pages. Illus. 1936.

PROCTOLOGY: A TREATISE ON THE MALFORMATIONS, INJURIES AND DISEASES OF THE RECTUM, ANUS AND PELVIC COLON. F. C. Yeomans. New York: D. Appleton-Century Co., 1936.

Fortunate indeed are those medical societies and physicians who have the second edition of Yeomans' Proctology in their libraries. The volume is very complete, covering all phases of proctology. A considerable number of outside references are included for those who wish to investigate certain phases of rectal disease not covered in the text. In this second edition, written after seven years, the advances in diagnosis and therapy have been carefully included. These have been cited in the preface, which saves time for anyone especially interested in the newer advances in proctology. The text is a valuable book, particularly as a reference for a practicing physician.

WALTER A. FANSLER, M.D.

THEORY AND PRACTICE OF PSYCHIATRY. William S. Sadler, M.D. 1231 pages. $10.00. St. Louis: The C. V. Mosby Company, 1936.

The text comprises 1155 pages and seventy-seven chapters. The author has been a prolific writer of popular pamphlets. This large book would embrace the field of psychiatry including its alpha and omega. Besides the subject matter commonly found in the

conventional text book on psychiatry, the author deals with much that is theoretical and, one might say, speculative. Because of the rapidly changing theories and empiricisms, we would expect such a treatise soon to lag behind the prevailing conceptions found in the current literature.

The author lists a general bibliography at the end of the text. There are thirty-four pages devoted to defining neurologic and psychiatric terms.

JOSEPH C. MICHAEL, M.D.

ABORTION, SPONTANEOUS AND INDUCED. F. J. Taussig, M.D., F.A.C.S. 536 pages. Illus. Price $7.50. St. Louis: C. V. Mosby Company, 1936.

This book should be considered one of the most outstanding publications of the times, being exceptionally exhaustive in its scope. It is very well written, easily read, and includes many excellent figures and drawings.

There is an article early in the book entitled, "Abortion in Animals," by Walter L. Williams, of Cornell University, which should prove of interest to obstetricians and gynecologists, for it is very apparent that we can learn much from veterinarian medicine and it may be that by closer study some problems in human medicine may be solved.

Taussig includes a chapter on the pathology of abortion which will be invaluable to those who study microscopically all aborted ova and feti. This is one side of the study of abortions which is often disregarded by the clinician and which, if taken up, will be of great aid in preventing future trouble. His chapter on treatment is most complete for he takes the type of treatment in vogue in most of the important countries and clinics of the world. Stress is laid on doing as little damage as possible to already injured tissue. Practically all forms of operative technic are described in both uncomplicated and complicated cases. The dangers of infected abortion are emphasized.

One of the most enjoyable parts to read was the chapter on missed abortion. The subject is so often disregarded in many books or else there is just a short paragraph assigned to it.

A section has been given to legalized abortion in the Soviet Union, much of the material being secured by first hand information from the author's visit to the Soviet States. Most of us have little or no knowledge in regard to this subject, and doctors to whom I have talked are surprised that there is such a thing as illegal abortion in that country. However, the Russians fully realize, as we all do, the harmful effects that can arise from therapeutic abortions and are trying to emphasize a better means—namely prevention, by means of educational methods.

The last few chapters of this book should be interesting to the Sociologist as well as to the Doctor because the economic, domestic, theological, ethical and legal aspects are discussed. One chapter deals with the extremely varying laws in the different states in this country.

This book should be in the library of every actively practicing physician for, to a great extent, the burden of a decreasing birth-rate rests upon him to overcome. Many condemn contraception, but if nearly every healthy conception could be carried through successfully, the rate of births in this country would not be cause for alarm. It is from this point of view that this book is especially valuable.

EUGENE M. KASPER, M.D.

ALLERGY OF THE NOSE AND PARANASAL SINUSES. French K. Hansel, M.D., M.S. Assistant Professor of Clinical Otolaryngology, Washington University School of Medicine; Fellow of the Association for the Study of Allergy, the Association of Resident and Ex-Resident Physicians of the Mayo Clinic, the American Laryngological, Rhinological and Otological Society, and the American Academy of Ophthalmology and Otolaryngology. 793 pages; illustrated. $10.00. St. Louis. C. V. Mosby Co. 1936.

It is unfortunate that this book bears a title which would tend to arouse the interest of only the rhinologist or allergist. The subject of allergy has developed so rapidly in the last five years that the internist, the gastro-enterologist, the dermatologist, the pediatrician, and even the general practitioner should "tune in" on it and its various manifestations. Here in this book is an admirable discussion of the various types of allergic disturbances. Written primarily for the otolaryngologist, the book is complete in that its early chapters lay a foundation of physiology, biochemistry, bacteriology and histology of the nose and sinuses upon which the subject of nasal allergic disturbances and its relation and association which sinus disease can logically be built. Details have not been slighted, and the sections on hay fever and elimination diets are especially complete.

The author's style is to be commended. There are copious references and quotations from the literature and he has been careful to discuss both sides of points which might be considered controversial.

It has been a distinct pleasure to review this work of Dr. Hansel's, and I unhesitatingly recommend it to anyone interested in the subject of allergy.

K. R. FAWCETT, M.D.